ARCHIVES OF OTOLARYNGOLOGY

VOLUME 43 MAY 1946 NUMBER 5

COPYRIGHT, 1946, BY THE AMERICAN MEDICAL ASSOCIATION

TREATMENT AND TRAINING OF THE HARD OF HEARING

A Program of Physical and Psychosocial Therapy

CAPTAIN FRANCIS L. LEDERER (MC)
AND
LIEUTENANT (jg) WILLIAM G. HARDY, H(S)
UNITED STATES NAVAL RESERVE

THE recent war has brought to the fore the urgent and essential need of adequate treatment for aural disabilities. Two immediate questions arise in the presentation of a program of aural rehabilitation:

1. Is the field extensive enough to warrant concerted action of all interested groups at a considerable expense of time and energy?

2. Is there a sufficient foundation of institutional experience with the treatment of large numbers of hard of hearing persons to enable interested groups to set up definitive procedures that are workable?

The time has passed when the first of these questions needs much debate. No one knows precisely how many deafened persons there are in this country, but, with the recent addition of a tremendous number of service personnel who have suffered acoustic trauma, it is safe to put the figure in the hundreds of thousands, and there is no indication that this estimate will diminish. As to the second question, the answer is an unequivocal affirmative. Both the Army and the Navy have set up programs of aural rehabilitation, which have already reached thousands of men and women and will continue to help thousands more in the months to come.

It is the thesis of this presentation that service experience with the reconditioning and reconstitution of the hard of hearing as distinguished from the congenitally deaf furnishes a pattern and methods that can be translated efficiently into civilian needs under the leadership of American otologists. There are presented an outline of the Naval Program of Aural Rehabilitation, some statistical findings to substantiate

From the Speech and Hearing Rehabilitation Unit, United States Naval Hospital, Philadelphia.

This article has been released by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions and views set forth are those of the writers and are not to be considered as reflecting the policies of the Navy Department.

the thesis and a prospectus of three organizational structures designed to meet the needs of various communities.

THE NAVAL PROGRAM OF AURAL REHABILITATION

Establishment.—A year and a half ago the Navy Department's Bureau of Medicine and Surgery established at the United States Naval Hospital in Philadelphia a national center for hearing rehabilitation. Here a special clinical and reeducational service is available to members of the Navy, the Marine Corps and the Coast Guard who have suffered handicapping losses of hearing. Developed to its present status under the leadership of Captain M. J. Aston (MC), U.S.N., medical officer in command, and Commodore R. A. Kern (MC), U.S.N.R., chief rehabilitation officer, the program has been organized under the direction of Captain F. L. Lederer (MC), U.S.N.R., and supervised by Lieutenant Commander H. Koepp-Baker, H(S), U.S.N.R., director of nonmedical services.

Incidentally, it is worth noting that the work of the Army and the Navy in hearing rehabilitation demonstrates parallel developments.

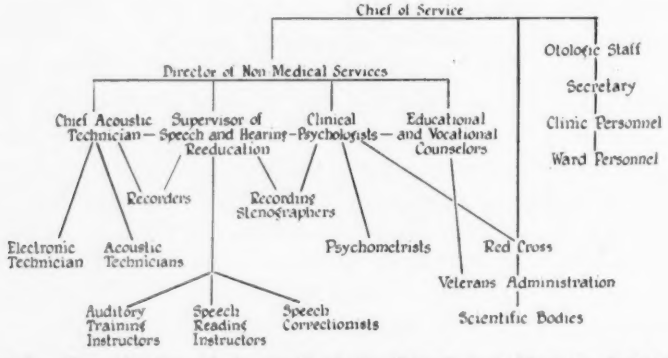

Chart 1.—Organization of the staff of the Speech and Hearing Rehabilitation Unit, United States Naval Hospital, Philadelphia.

There has been constant liaison between the two branches of service in all aspects of this special rehabilitative task; each finds itself working in modalities used by the other, and there is a high degree of conformity in matters of etiology and in statistical findings.

Staff Personnel.—The Navy brought together in Philadelphia a group of specialists to concentrate their attention on the treatment and training of speech and hearing rehabilitees. The professional fields represented include otology, psychology, speech reading, speech correction, auditory training, audiometry and acoustics. Another group, composed of occupational therapeutists, specialists in educational services and prevocational training, physical training instructors and medicosocial workers of the American Red Cross, has been assigned collateral duties in the program. (See chart 1.)

It should be borne in mind that the rehabilitation of the person with a hearing disability is primarily a nonmedical operation. After initial examination, diagnosis and treatment, the great majority of rehabilitees require relatively little medical attention. Moreover, the scope of hearing rehabilitation is broad; any training that strikes so deeply at the core of psychosocial behavior presents many facets, each of which requires expert attention. Because of the size of the case load, it has been necessary for the staff personnel to specialize their duties. A smaller clinic than that required by the naval program could be adequately manned with fewer trained specialists by grouping the assignments.

In the final analysis in hearing rehabilitation the size and the efficiency of the staff of technicians and teachers are conditioned by the character and the extent of the physical space and the equipment. Yet the technical operations—the quantification of hearing loss and the evaluation, selection and fitting of hearing aids—limit the number of patients that can be handled adequately within any training period, although classes in speech reading and auditory training can be expanded within reason.

Standards of Admission.—Naval and Marine Corps personnel to be admitted to the retraining program must have a hearing loss of at least 30 decibels in the better ear within the conversational range. By definition such a loss constitutes a hearing disability that is a handicap. In general, unilateral deafness is not considered a serious handicap. The level of 30 decibels is a reasonable standard, as such a loss is usually subjectively noticeable; conversation in the presence of noise entails confusion and uncertainty. It is our conviction that performance in speech reception must receive primary consideration among the various implications of faulty auditory acuity. We are committed to the retraining effort necessary to bring communicative intelligibility as close as possible to the level of normal performance. It goes almost without saying that allowances are made in terms of individual variations minimally above or below the 30 decibel impairment.

Patient Load.—At its peak in midsummer of 1945 the patient load at the center in Philadelphia averaged between 600 and 700. Patients are held for a period of five to six weeks in a highly specialized retraining program under the guidance of an all-military personnel. With but few exceptions our "graduates" have received sufficient fundamental instruction in speech-reading skills and in the use of a hearing aid to enable them to adjust to the requirements of civilian existence and to assure them of a fair chance for social and economic sufficiency.

Source of Patients.—Patients are transferred to Philadelphia from naval stations the world over by means of a "medical survey," a complete

record of the history, the findings and the recommendations pertaining to the individual's case. When a man is on duty outside the continental limits of the United States, several surveys may be required, so that, island by island, he moves through a chain of naval medical establishments to the rehabilitation center. The Bureau of Medicine and Surgery is well aware of the enervating effects of prolonged hospitalization, and every effort is made to speed up the process of transfer.

Philosophy and General Approach of the Retraining Program.—What constitutes a person's rehabilitation? What is he being rehabilitated for? The Surgeon General of the Navy, Vice Admiral Ross T. McIntire, has defined rehabilitation as meaning "all activities and services which may be required to supplement the ordinary or usual therapeutic procedures in order to achieve maximum adjustment of the individual patient either for further military service or for a return to civil life with the least possible handicap from his disability." Aural rehabilitation in these terms involves some broad considerations that relate to the core of a person's psychosocial behavior. Is a man with a hearing loss different from what he was before he became deafened? If so, to what extent, and what should be done about it—what *can* be done about it? These are the questions confronted daily by those responsible for the successful operation of speech and hearing rehabilitation. Their answers reveal many factors concerning the psychology of deafness and the retraining process established for the rehabilitees.

The fundamental idea of such a program is its centralization in the rehabilitee's personality. The goal is the reconstitution of the *total* person, and every aspect of the program relates to the adjustment of the person's behavior in terms of the exigencies of his aural disability. We are not solely concerned with his ability to read speech through muscular cues, nor with his ability to get and hold a job. We are concerned with the psychosocial function of the whole person. We concentrate on communicative activities simply because they furnish the core of this function.

There are at least three principal elements which characterize this approach to the rehabilitee's problems. The first has been expressed in a simple aphorism: "A man is more important than his ears." Specialists in hearing and speech proceed in the belief that they are under obligation to treat the total personality. In the second place is their recognition of the need to arrest deteriorating characteristics and to do everything possible to help restore the subject's complete existence: The rehabilitee must learn to become not a handicapped person who travels alone, dependent and frustrated, but a "person who wears a hearing aid," who is in communication with his fellows and who is as capable of economic and social efficiency within the limits of his potentialities as any other man. Third is the need to help an aural rehabilitee

expand certain of his capabilities in order that he may compensate for a hearing loss in a complex socioeconomic existence.

All of the varied activities of the program serve cumulatively to build up a pattern of successful performance for the human being. They serve to prevent the deterioration of self respect, which remains the principal threat of a sensory handicap. Through his controlled existence at the retraining center the rehabilitee receives concrete evidence of his ability to live normally; he can look forward to a measure of security in a highly competitive world, the kind of security enjoyed by a man who does his job well. Essentially, then, so far as the psychology of deafness is concerned, a proper functioning speech and hearing program is an individualized course of mental hygiene; it is aimed to help a person gain and maintain self confidence, to give him the foundations of self support.

Manifestly, a program of rehabilitation that is patient centered must undertake two essential steps toward the goal of balanced behavior of the rehabilitee. The first is positive: the development of new abilities and skills, new tools to achieve efficient and successful behavior and adjustment. As positive therapy, moderate proficiency in speech reading, training in sound discrimination and supervised use of a hearing aid are fundamental elements of the patient's equipment for normal behavior. The second is negative: the understanding and nullification of the adverse effects that are inevitable by-products of any serious interference with communicative habits. The rehabilitee must learn new ways of getting along in a world that is dependent on communication. If he wears a hearing aid, he must learn to think and act like *a person who wears a hearing aid*, not like a person who is perfectly normal but cannot hear well. He must prepare himself to meet the thoughtless attempts of others to communicate with him. He must learn to be quite tolerant of his more fortunate fellows. On the other hand, the person who is deprived of the free use of language tends to turn inward, to become depressed, unobjective, negative in his attitude toward himself and his fellows. These tendencies must be understood and nullified; otherwise, a great part of what a rehabilitee may learn is rendered useless.

To help him keep his attention centered healthfully and successfully on the elements of normal living, much must be known about him, and expert help is needed to steer him in the right path. While it is too soon to assemble the facts about discharged veterans with hearing losses, available evidence indicates reasonably good adjustment to the business of getting along in a new world.

Processing the Patient.—When a man is admitted to the hospital, he receives a thorough physical examination and is then "processed"

for the Aural Rehabilitation Service. He is examined by an otologist who obtains a complete case history of the principal symptom, deafness. He then receives a battery of functional hearing tests, including conversational and whispered voice, Weber, Rinne, audiometric and speech reception measurements. Lombard and Stenger tests are employed when simulation is suspected.

These tests are considered valid to the extent to which they are used. There is little question that the conversational voice test is so variable as to be essentially meaningless; it produces only the roughest kind of estimate, which is serviceable only for the early stages of screening. It is retained in the process at Philadelphia only because it remains a traditional requirement in the eyes of various boards of review; it still remains the basis of the adjudication of disability claims in the Veterans Bureau. The whisper test, too, is retained only for screening purposes. Whispered voice seems to be valid mainly for measuring high frequency loss; its primary clinical virtue appears to be its simplicity and the ease with which it can be administered. The whisper test in Philadelphia is conducted with the utmost care, with the use of residual air, in order to produce objective control. Masking is generally recommended in the appraisal of the hearing ear.

Examination with tuning forks is believed to have clinical virtues that are valuable. The forks, properly used, demonstrably aid the interpretation of biographic data and help to clarify the problems of accurate diagnosis. Audiometry, carefully conducted with regularly calibrated instruments, is considered an accurate measurement of hearing thresholds. The classic six octave range is employed, with every measure that reasonably insures against elements of fatigue and inattention. Nor is audiometry limited to the use of discrete frequencies. A correlative part of the examination is the measurement of speech reception in a free field. This is performed with a two channel instrument developed under the auspices of the National Defense Research Council and perfected in the acoustic laboratory of the naval hospital. With this so-called "hearing aid evaluator" the patient's threshold of speech reception can be measured accurately within 1 decibel. Together, these tests for thresholds are considered fully adequate to determine the extent of hearing loss for all practical purposes.

When the quantification of hearing loss has been achieved, the patient meets the medicosocial workers of the American Red Cross who are attached to the department, a specialist in educational services and prevocational training, and a departmental psychologist. The Red Cross representatives help him become oriented to the hospital situation, supervise social and recreational activities, and provide help and counsel in a diversified range of familial problems. Because of the psychologic aspects of a loss of hearing, this is an invaluable adjunct to the retrain-

ing program. The specialist in prevocational counseling helps to determine an appropriate future for the hard of hearing person and furnishes advice and contacts for a program of work therapy which can be followed while the patient is in residence at the center. The departmental psychologist, thoroughly acquainted with every aspect of the rehabilitation program, establishes rapport at the outset and illuminates problems of adjustment to the loss of hearing. This particular interview is exhaustive and is considered the clinical key to the reeducational process.

Each patient is interviewed by an instructor in speech reading, who ascertains his natural ability as a speech reader in a conversational situation and grades him accordingly. If a hearing aid is indicated, as is true in most cases, an impression is made at once for an individual, "tailor made" ear mold. Finally, prior to the initial day of classes, the incoming patients meet as a group with the director of nonmedical services, who lectures on problems of mental hygiene and the psychology of deafness.

Once all the data on each entering patient are assembled and made available to the staff, the patient is assigned to classes and the retraining course is under way. The hearing rehabilitees are quartered in special wards where they are out of direct contact with other types of disabilities. Sick call is held daily by the ward medical officer, who handles all definitive treatment except what necessitates consultation with and referral to other departments. It is usually only a matter of a few days before the patient finds his niche in the routine.

Group Therapy.—The importance of group therapy in the training of aural rehabilitees can hardly be overemphasized. From the earliest stages of a patient's indoctrination until his discharge from the center he meets and shares with a group of his fellows the interests and concerns common to all. Throughout their stay in the program the rehabilitees are brought together twice each week to take part in generalized discussions of their problems. Several meetings are carried on under the leadership of a departmental psychologist who trains the men in mental hygiene and in facing the problems of the deaf. Others consist of lectures by staff otologists on the hearing mechanism and the pathologic changes that may occur in it; special attention is paid to the causes of hearing loss in the Navy. Later meetings afford opportunities to discuss the physical attributes of hearing aids and their care and use in terms of typical vocational problems.

The men are given ample opportunity to ask questions and to satisfy their own particular needs of adjustment. The results of these sessions have been salutary; the results are of course difficult to measure objectively, but we are satisfied that the group meetings serve to

complement the individualized treatment and provide the necessary social elements of sound therapy.

Medical Services.—It should be fairly obvious that hearing rehabilitation is largely a nonmedical matter. The system in operation in Philadelphia is one of fine coordination and implies the closest cooperation between medical and nonmedical officers. Administratively the Speech and Hearing Rehabilitation Unit is organized under the Eye, Ear, Nose and Throat Service, and both the chief of service and the ward medical officer assigned to hearing rehabilitation assume responsibility for the physical fitness of each patient and for his status under official medical survey for hearing disability.

An otolaryngologic clinic is operated in the building which houses the retraining work, and all routine medical and surgical procedures are carried out. An otologist handles otoscopic examinations and all definitive treatments; technicians administer the functional tests under his supervision. Treatment for systemic conditions is undertaken by the ward medical officer whenever practicable; in cases of contagion or complication, transferral to other special medical services is arranged. There are, of course, available to the aural rehabilitee all the routine laboratory procedures and interdepartmental consultations that a major hospital affords.

In some instances, surgical treatment by fenestration of the labyrinth is indicated. A recent directive establishes the United States Naval Hospital, Bethesda, Md., as the center for this treatment. The following requirements are operative: (1) a diagnosis of otosclerosis; (2) a bilateral conduction type hearing loss averaging at least 40 decibels through the frequency range from 256 to 2,048 double vibrations per second; (3) normal or very slightly reduced bone conduction through the same frequency range; (4) the patient's election of the treatment. This procedure is limited to naval personnel on active duty.

The Reeducational Program.—The reeducational program for aural rehabilitees has three elements—speech reading, auditory training and speech training. The degree, the type and the duration of the hearing loss and the prognosis for each patient should control the emphasis put on each of these elements. In general terms, the work in course is designed for a period of four to six weeks, with class time scheduled for five hours each week in both speech reading and auditory training. This schedule is open to modification, however, according to demonstrated individual needs. The nature and the degree of the hearing loss determine in large measure the need for the rehabilitee to develop particular skill in speech reading or to undertake particular training in the use of residual hearing. For general retraining purposes the

patients are classified in three categories: those with marginal, those with moderately severe and those with profound hearing losses.

The question of fitting a hearing aid early or late in the reeducational process has been debated warmly by aural specialists. We have adopted the policy of fitting every patient with an aid who can profit from its use just as early as possible. The gains in confidence and the ease of communicative and social adjustment more than compensate, we believe, for any lag of effort to master speech reading.

An introduction to English phonetics furnishes a working foundation for the retraining task. This is a simple study of speech articulation which enables a patient to determine where, and to some extent how, sounds are made. Once speech sounds are localized, he practices to learn how they feel as he talks, and this experience develops his kinesthetic awareness, a direct step toward a grasp of the basic idea of speech reading as it is taught in the naval program,

It is believed that the Navy's training methods offer a genuine contribution to the work being done with the hard of hearing. Many of the ideas suggested here must be credited to Lieutenant (jg) Miriam D. Pauls, H(W), U.S.N.R., supervisor of reeducation, whose technical acumen and personal enthusiam have contributed immeasurably to the development of this aspect of the program.

Speech Reading.—The technics of instruction in speech reading are by no means new, yet they are in a somewhat liquid state. Modern psychologic research has brought about some vital changes in older ways of approaching the problem. Accordingly, the Navy's program is partially experimental, and instructors hold themselves ready at all times to adapt new ideas and special technics to the task at hand. If any one system can be said to furnish the core of our approach, it is the Jena method.

The Jena method invites a close correlation among speech, speech reading and auditory training of residual hearing. Its fundamental precept is that the kinesthetic, or "feeling," form of communication is the only form that is complete for everybody under all circumstances. Thus, this system invites special attention to the muscular cues in association with whatever audible and visible percepts are available: patients are taught to feel, hear and see simultaneously. (See chart 2.)

That is to say, the naval rehabilitee is taught not lip but speech reading. Only about a third of the sounds of English are visible. The patient spends hours talking simultaneously with the instructor, while he sees, feels and hears the general pattern of expression. Then, in an effort to recall all the sensations of the pattern when he spoke it aloud, he watches the instructor while the same material is repeated. The next step is silent practice with the instructor, who preferably addresses the group from a position behind a soundproofed glass partition. In

this situation, the patient works without sound, being entirely dependent for his reception of communication on visual cues and on his ability to recall and interpret the kinesthetic response.

The average person acquires this technic of reading speech with considerable ease. First lessons are simple; they include syllable drills and simple series of words which incorporate a "psychologic set," such as counting, enumerating the days of the week, various colors and the like. Gradually the difficulty of the material is increased, and various complex elements are introduced. The student works with grammatical forms and with typical conversational situations. Dramatic skills are employed to acquaint him with different life situations, and it is recommended that motion pictures be utilized to help the patient gain further facility in recognizing patterns of speech and in building a vocabulary. Practice with "regular run" motion pictures emphasizing close-ups is

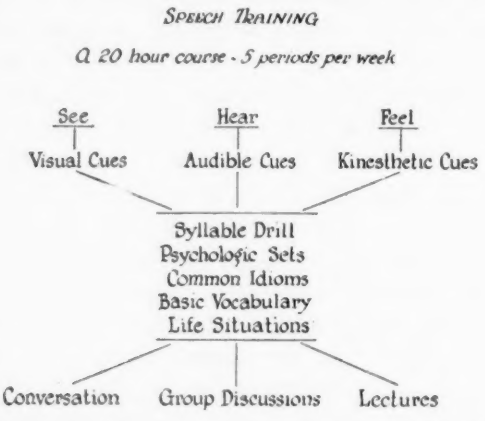

Chart 2.—Schematic outline of the course in speech reading of the Speech and Hearing Rehabilitation Unit, United States Naval Hospital, Philadelphia.

good, and there are varieties of specially constructed training films available.

Such technics employed with the newly deafened are quite different from those used with the congenitally deaf. Our rehabilitees come to the center as mature persons who have had serviceable hearing most of their lives. Patient and instructor are concerned primarily with responses to life situations, and the men are retrained accordingly. A similar state of affairs obtains, it seems to us, in considering retraining measures for civilians who have become hard of hearing and instituting preventive measures against the development of hearing disability. The hard of hearing person has a background of language and communication which should be appropriately capitalized.

An acquaintance with the sound values of language having been acquired, the classroom sessions move directly into conversational

material. This sustains a lively, alert situation between instructor and students. Ideas, rather than casually selected, stereotyped phrases, furnish the subject matter. A basic vocabulary of common words and idioms is part of the daily drill, so that a man soon learns to recognize these terms with ease. Moreover, in the advanced stages of training, a wide variety of practice situations is offered: lectures, group discussions and the like. These are presented in anticipation of the situations in which the patients will find themselves on discharge from the center.

Homogeneous groups are set up according to the patients' demonstrated natural ability for speech reading. (Actually, there are few people who do not read speech to a certain extent.) Small groups, from six to ten, work best—far better than one person receiving individual instruction. It is our conviction that in speech reading group instruction pays excellent dividends. It introduces healthy competition, variety of subjects and constant exposure to several patterns of speech. Individual instruction is provided when it is demonstrated that a man is not making good progress with the group; but he is returned to the group as soon as possible.

The same groups are not maintained throughout the course. Each group remains with an instructor for about a week, then is assigned to a new instructor. Thus, in the training period a man has experience with four different teachers, and by the time he is ready for his final proficiency test, a requirement for his completion of the program, he has been exposed to a fair variety of problems in speech reading.

Teachers are encouraged to keep their instructional materials fluid, with frequent revision of content material and with incorporation of new ideas as they are demonstrated to be good. Such a procedure tends to keep the instructor alert to a task which otherwise is apt to become routine and dull.

Auditory Training.—For good results in the use of a hearing aid, auditory training is usually necessary. This is preferably carried on in conjunction with speech reading and entails practice in listening to amplified sound through both group and individual hearing aids in carefully controlled situations. The men meet in groups of eight to ten five hours each week for a course of instruction that has been developed from four closely related aspects of aural rehabilitation. (See chart 3.)

In charge of auditory training at Philadelphia is Lieutenant (jg) Harriet L. Haskins, H(W), U.S.N.R., who is assisted by a highly trained group of officers and enlisted personnel, including several professional speech pathologists.

The Navy's work in auditory training has amounted to a genuine pioneering effort; there was almost no precedent to follow, and the

precise nature of the psychophysiologic problems involved was relatively undetermined. We were convinced, however, that a person could not achieve the optimum use of a hearing aid without something more than a few kindly words of advice. In a twelve month period of experiment, adaptation and adjustment we have learned a good many lessons about the training of residual hearing with the use of a hearing aid.

Early in the course patients are given a sound discrimination test without aids. This is followed in a few days by another test with a group hearing aid. Other tests are spaced through the four weeks of the course to furnish both instructors and rehabilitees with reasonably accurate objective data on hearing performance in various circumstances, including the identification of situational sounds and noises, performance in conversational backgrounds and the like. Some of the standard tests in sound discrimination have been tried and found unsatisfactory,

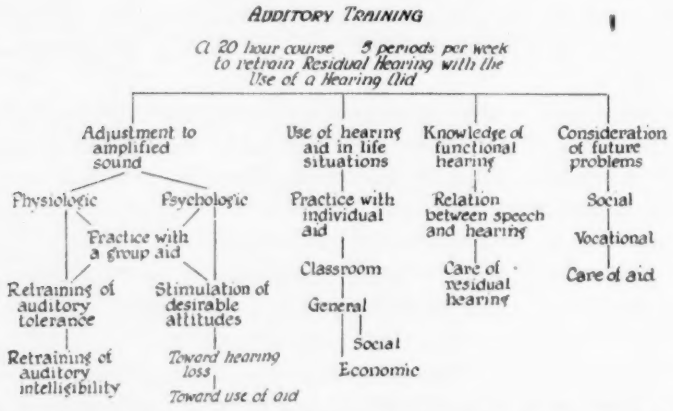

Chart 3.—Schematic outline of the course in auditory training of the Speech and Hearing Rehabilitation Unit, United States Naval Hospital, Philadelphia.

particularly those featuring the ability of the hard of hearing person to distinguish among various high frequency consonants. The fact seems to be that hearing aids as we now know them will not pass undistorted frequencies above the level of 4,000 cycles per second and that even the most persistent drill with words containing high tonal complexes beyond the range of amplification given by a hearing aid accomplishes little good. The instructors in auditory training concentrate most of a group's attention, therefore, on broad phrases which represent a normal speech stream.

Although it is not feasible to group the men precisely according to their degrees of hearing loss, those with either marginal or profound losses receive the special attention they need. The first group, those with marginal losses, requires special emphasis on speech training and

speech reading, for many of the men cannot accept the amplification of a hearing aid; the second group, those with profound losses, many of whom hear only a few discrete frequencies and have a speech reception threshold indicating 90 or more decibels loss, need special work in the recognition of gross sounds, rhythm patterns and vowel tones before they are ready to interpret sounds well enough to use amplification profitably.

For the rest of the aural patients, the vast majority of our rehabilitees, the inital stage of auditory training is concerned with the ability to listen comfortably to amplified sound. A multiple hearing aid is employed that brings the men vocal and instrumental music, speech and sound effects either directly from the output of a record player or through a speaker—microphonic pickup that allows the introduction of ambient noise and thus provides more normal hearing situations. An attempt is made to provide the men with unilateral hearing, only one ear phone being used, in a fashion consonant with the effect they will get from their own individual aids. As a matter of fact, it has been determined that the appropriate fitting of an individual aid is made considerably easier by these early listening experiences on the group aid, and the fitting schedule is regulated accordingly.

There follows an introduction to speech, first under quiet circumstances, then graduated to include typical communicative conditions. There is ample opportunity for the men to adjust to various types of voices. Then, when their own aids are available, within the second week of instruction, they receive extensive practice in general conversation and group discussion. The masking effect of noise is transmitted through a loudspeaker while conversations are carried on.

Meantime, individual tolerance is carefully noted. Every effort is made to avoid exposing a patient too early to an undue amount of sound pressure; by the same token, every effort is made to encourage him gradually to raise his level of tolerance. In some instances of nerve deafness, particularly those in which the hearing losses have extended over a considerable period, this is not readily accomplished. But with few exceptions it has been found that tolerance can be educated to a significant extent, with all the resultant improvement in auditory efficiency.

General speech training is introduced early in the course. The groups hear lectures and perform exercises with the various controls of speech—pitch, rate, resonance, quality and force—and attention is drawn to the problems of the "deaf voice" and allied vocal deteriorations that are apt to follow from a loss of hearing. This aspect of the work is of course closely related to the kinesthesia of speech reading, for each man with a hearing aid must learn anew the cues necessary to help him with vocal discrimination.

The men are encouraged to try their aids in a variety of circumstances; they report on their progress and discuss particular problems that arise as they move about the hospital and in the city of Philadelphia. They are urged to experiment with different ways of wearing an aid and to report their success or failure. Here are included the problems of clothing noise, cord static, body-baffle and the like. The group is often able to profit from each member's ingenuity, and numerous practical suggestions have been offered. The total effect of this kind of activity suggests a second major aspect of auditory training: to give the patient concentrated experience in adapting himself to his hearing aid, in learning to adjust to it in various communicative circumstances, in discovering early what an aid can and cannot do for him and in meeting his particular hearing problems with an attitude of alert willingness to learn how to adjust.

The men are trained in the use of a telephone while wearing an aid, and if a person is profoundly deafened, he is expected to use the center's special instrument that contains a hearing booster.

These objectives of auditory training entail a third aspect of the work: to train the rehabilitee to understand his own hearing problems. He should become expert in his handicap. Within the first three weeks of the course each man's audiogram is analyzed and discussed. Special consideration is given to his medical diagnosis, the history of the loss, the presence of tinnitus, the condition of the tympanic membranes and his threshold of speech reception. For the most part, the men are vitally interested in all the diverse aspects of their disability and have many questions to ask. A patient knows he may ask for and receive advice on what troubles him most, and he knows he is not alone in his difficulties. The psychotherapy of such an approach has more than a little value.

The fourth objective of auditory training is an outgrowth of what has just been described. In the closing sessions of the course, after the patients have begun to adjust well to their aids and are fairly adept at speech reading, some time is devoted to the problems of conserving the hearing of each and obtaining service for his aid after he has been discharged. The effects of various types of jobs are considered, and an effort is made to foresee the kinds of problems that will arise in the man's community. These run the gamut from the need for regular otologic examinations to queries about means of procuring fresh batteries and new cords. For the most part, these topics fall outside of immediate naval jurisdiction, and to obtain accurate information men are referred to the district representative of the Veterans Administration, who is accessible through various local organizations. The naval program can help the rehabilitee to foresee many of his problems; how they will be met institutionally is the concern of civilian agencies. From

reports received to date in follow-up questionnaires, we judge the men are having particular difficulty in securing fresh batteries and adequate repair service.

In sum, the course of auditory training involves a complete reeducation of the person's hearing habits. He is expected to master the changes and the nuances of connected discourse so far as this may be accomplished. He is acquainted with his own hearing problems and with some of their solutions, and he learns to adapt himself to his own hearing aid. Finally, he is aware, at least to a considerable extent, of his future as "a person who wears a hearing aid."

Speech Training and Correction.—Daily exercise in articulation drills and in discrimination of sounds forms an important part of the training in the reading of speech and the use of residual hearing. Special attention is paid to the patients' articulatory habits in an effort to tone up their speech patterns. The goal of this phase of the rehabilitative effort is not to put the emphasis on any special pattern of speech but to foster clear serviceable speech and to stress the relations between speech and hearing. The patients must learn discrimination in the production of their own speech and know how to guard against the deterioration of speech that so often accompanies a hearing disability.

Most of our rehabilitees show no evidence of serious speech deterioration; their disability is so recent that their normal habits of speech still persist. For them, this work in speech is insurance against the future. Others, particularly those who had hearing losses prior to enlistment or induction, require definitive correction of speech. And those with profound losses require special attention to speech problems, with the emphasis put on kinesthetic cues. All those with special difficulties due to speech disorder receive the individual treatment that is indicated.

Audiometry and the Fitting of Hearing Aids.—The quantification of hearing losses and the work of evaluating, selecting and fitting hearing aids are under the direction of Lieutenant (jg) Eva A. Thompson, H(W), U.S.N.R. For many years assistant to the late Walter Hughson, both at the Johns Hopkins Medical College and at the Otological Research Laboratory, Abington, Pa., Miss Thompson first worked in this capacity for the Navy as a civilian, then accepted a commission in the United States Naval Reserve.

Those tests which determine the type of hearing and establish the degree of loss in qualified terms are of primary importance. They furnish conclusive data by which to determine the usefulness of a hearing aid; or, if an aid is contraindicated, these tests suggest the pattern of retraining necessary for a patient.

For some time now the audiometer has been an accepted standard instrument for measuring the acuity and the range of hearing. Audio-

metric tests for the hard of hearing are best given in a sound-treated room and must be conducted by an experienced technician. A sound-proofed room is not always necessary; yet competent audiometry, competent, that is, to measure hearing accurately and present trustworthy data for diagnosis, must be conducted in a controlled situation. Granted that an ambient noise level of 35 decibels is unimportant in testing a hearing loss of 50 decibels, yet so many variable factors must be considered in measuring both discrete frequencies and speech reception in a free field, using both air and bone conduction, with and without masking, that the control of the testing apparatus is imperative. It is not sufficient to set up an audiometer in a corner of an office. To the variables of ambient noise and standing waves must be added the factors of distraction and fatigue. Disregard of all these features reduces the process to the ridiculous. We have had too much experience in the Navy with audiometry carried out in a haphazard fashion with instruments that have not been calibrated for months.

So, too, with the audiometrist. One does not do good audiometry without guidance, without experience and without knowledge of the technics available and the problems to be met. It is not good enough to assign the job part-time to a medical secretary who knows no more about audiometric testing than to snap switches and turn dials. Granted that there are varying technics available; granted further that authorities do not agree on the proper technic. A capable audiometrist should be acquainted with three or four good technics and should know enough to vary his procedure with the patient. Every aspect of work with the hard of hearing suggests the need for respecting the psychosocial behavior of the patient, and good audiometry is no exception. With due care the audiogram is a graphic representation of the patient's hearing loss.

Supplemental to this test is the measurement of the threshold of speech reception. Indeed, there is a valid argument in the idea that the speech reception test is the most valuable single measurement of a person's hearing acuity. It can be made accurate to 1 decibel and offers a conclusive check on a patient's ability to function in a speech environment.

The speech reception test requires two rooms, one a sound-deadened room in which the patient is seated, the other a quiet booth for the technician. The necessary apparatus consists of a microphone, an amplifier, a volume indicator, an attenuator and a loudspeaker, together with a communication system between the rooms. Spoken voice, usually consisting of test sentences, is delivered to the patient by a reader. The patient is seated at a fixed distance from the loudspeaker, and the intensity of the spoken voice is controlled by an attenuator. Considerable intensity is used at first; this is gradually reduced until the threshold

is established. Intensity as great as 80 decibels, considerably more than average loud speech, may be used without overloading the equipment.

The type of audiogram, the residual hearing in speech reception, the results of the otoscopic examination, the history of tinnitus and other pertinent data, all contribute to the selection of the hearing aids to be tested for the rehabilitee. Individual hearing losses may vary in many ways from the classic patterns of hearing loss; no two persons are precisely alike. Moreover, the acoustic technician conducting the tests must be thoroughly acquainted with the properties and special characteristics of the aids at hand, for many are designed for specific types of hearing loss. The Council on Physical Medicine of the American Medical Association has established the minimum requirements for acceptable hearing aids. In cooperation with the Army and the Navy, the National Defense Research Council has set up procedures for testing and evaluating various aids under stringent laboratory conditions, and furnishes information for comparative studies on their performance. An aid which meets requirements of the Council is considered satisfactory, and the service organizations have made excellent use of the National Defense Research Council data, which, when their significance as restricted material no longer obtains, should become available for distribution to interested persons. This information should have a far reaching effect on the attitudes of hearing aid manufacturers, who will naturally want their instruments to meet the highest possible standards, and, further, will inevitably influence competition in the hearing aid industry. Such objective data should serve to protect the interests of many members of the industry, whose organizations spend considerable time and money in laboratory investigation. The influence of the findings of the National Defense Research Council is already apparent in the enormous strides taken within the past year in improving hearing aids.

The selection of the proper ear to be fitted is determined according to various data on the amount of loss, any significant disparity between the ears, whether or not bilateral hearing can result, and the like. On the basis of all available medical and acoustic information about a patient, the chief acoustic technician makes a preliminary selection of three or four aids. This initial judgment is supported by a screening test wherein the patient uses these aids under normal conditions and has an opportunity to reject those whose performance is unsatisfactory. These subjective judgments are ordinarily made in terms of quality and annoyance. Thereafter, the final choice of an aid is determined by speech reception tests in a sound-deadened room. The testing procedure is continued until both technician and patient are satisfied as to details of performance on all counts. No fit is considered

satisfactory unless the aid produces a minimal gain in speech reception of 30 decibels (or brings the hearing into the normal range) and is comfortable in every respect.

Incidentally, although members of the staff were at first dubious about the successful use of an aid if the rehabilitee had a hearing loss of greater than 80 decibels, experience in fitting persons with profound loss has been salutary. Many patients with hearing acuity measurable only at one or two middle frequencies have been fitted with aids that bring some sound cues to them and thus enable them, with careful auditory training and with skill in speech reading, to get along reasonably well. This has happened too frequently to be exceptional.

The selection and fitting of the proper hearing aid is a process that is vital to the patient's future behavior. Contributory to satisfactory performance is the fit of the ear mold; each is fabricated individually, and checked and rechecked. Moreover, the process of fitting is based on evidence that no single hearing aid is outstanding in performance. Each case offers its unique problems. Even the audiogram furnishes insufficient evidence for the selection of the proper aid for the patient, and there is no evidence available at present that the consideration of matching or mirroring the audiometric curve should set aside all other psychophysiologic factors; only the speech reception tests given under various conditions can check an aid's performance and utility. Fitting the aid is a highly individual process, and a choice is made only when an aid satisfies the severest subjective and objective standards.

Physical Facilities.—One may well believe that the type of rehabilitative service outlined here places heavy requirements on the physical plant that houses such a unit. Fully realizing the implications of its extensive program for speech and hearing rehabilitation, the Navy laid its plans eighteen months ago for expanded quarters and suitable equipment. Since that time, with the professional counsel and direction of Lieutenant Commander Herbert Koepp-Baker, H(S), U.S.N.R., our director of nonmedical services, the work of installation has gone forward to completion. The new unit, designed especially for aural rehabilitation and erected as an integral part of the facilities to house the deafened personnel, is as thorough an undertaking architecturally and acoustically as imagination and sound understanding of needs can make it.

The availability of adequate physical facilities and acoustic equipment for aural rehabilitation cannot be overemphasized. It is not enough to sound-treat a room here and there, and proceed with an audiometer and a record player. "Hopkin's Log" is not quite sufficient for the rehabilitation of the sensorially handicapped. The naval unit has

been designed and constructed from the foundations up with the particular services in mind to which the unit is dedicated.

Every step of construction from the initial rough drawings to the finished product has been meticulously executed for optimal therapeutic results. And with the advice of the National Defense Research Council, naval planners have selected the best equipment available.

Laid out on one floor (a concrete-poured foundation houses only the air-conditioning equipment), the unit contains a closely correlated set of facilities. There are nine classrooms for speech reading, each equipped with a group hearing aid and a built-in glass booth that makes possible instructional demonstration with full voice. Several rooms have been set aside for otologic and psychologic examinations. A large lecture room, equipped with radio, phonograph, balanced loudspeakers and a motion picture projector, serves a variety of purposes in group therapy; close at hand is a record office and a generous space furnished with built-in lockers to house the library of hearing aids. There is one large classroom, with special equipment for telephonic communication, and several smaller ones for auditory training. These contain facilities for every phase of retraining in the identification and discrimination of sounds.

In a specially constructed wing, actually structurally separated from the larger building, is housed the equipment for testing and research. Here are windowless rooms for audiometry and special quarters for correction of speech and acoustic research. Each of two acoustic laboratories has a quiet booth, off which are two sound-deadened rooms floated in the building's structure and securely insulated from both internal and external building vibrations. The whole unit is air conditioned by a system supplied with special sound dampers and laid out for minimal acoustic interference. So far as it is humanly possible to foresee our needs, every architectural detail which promotes absolute control of acoustic values—and thus of therapeutic objectives—has received full and thorough attention.

CORRELATED CONSIDERATIONS

Hearing Loss in the Navy.—At the present time almost 3,000 patients have been cared for at the center in Philadelphia. The accompanying table presents a statistical analysis of 2,500 of these cases and reveals the typical disorders met in the Aural Rehabilitation Program.

Inasmuch as a great part of our information is gained perforce from the patient's own statements regarding his disability, the determination of etiologic factors often entails considerable study. True, we employ roentgenograms to identify possible pneumatization of the mastoid process and to detect chronic suppuration when the restriction of cellular

conformation indicates previous disease. We believe, however, that there is no more error in the study of our statistics than obtains in those from any clinic where a certain amount of dependence must be placed on the taking of case histories.

Then, too, in another connotation the two major categories of the table assume particular emphasis. For we have to determine not only the service connection of deafness but the extent of aggravation that has been sustained beyond the preservice disability reported by the patient. This entails even more thorough study of possible etiologic factors. In evaluating the biography of a patient the Board of Medical Survey gives every reasonable benefit of doubt to the claim that the hearing impairment has been aggravated by service in the Navy. To date about 93 per cent of the patients with a hearing defect involving

Analysis of Twenty-Five Hundred Cases of Deafened Service Personnel

	Number	Per Cent
Deafness Existing Prior to Enlistment or Induction:		
Chronic, progressive types	497	19.88
Nerve types	378	15.12
Chronic otitis media	110	4.40
Totals	985	39.40
Deafness Incurred in Service—Deafness caused by:		
Heavy fire	664	26.56
Epidemic meningitis	28	1.12
Mumps	6	.24
Trauma of skull	27	1.08
Whooping cough	1	.04
Machine noises	54	2.16
Underwater concussion	8	.32
Chronic suppuration	90	3.60
Nerve deafness	379	15.16
Chronic, progressive deafness	258	10.32
Totals	1,515	60.60

impairment existing prior to enlistment or induction have been recorded as having a condition related to the service through aggravation. We feel that in the service under the stress of various duties all ears took abuse beyond that of the ordinary conditions of civilian life.

Our patients have had their share of infections of the upper respiratory tract. They have been exposed to certain vicissitudes of climate which no doubt played a role in aggravating even preservice aural conditions. The majority of the members of this group, varying in age from 18 to 64 years, suffered no dietary restrictions; so factors of malnutrition do not enter into the etiologic picture. Exceptions are a few of those who were Japanese prisoners of war; they ordinarily have a history of beriberi resulting in auditory loss, sometimes in association with blindness. It is worthy of note that naval personnel are generally exposed to loud engine noises, on aircraft carriers, when working with Diesel-operated vehicles, in engine rooms and the like.

A primary cause of acoustic trauma is, of course, heavy firing. The result is classically nerve deafness, with the onset often brought about by exposure to a single salvo or an unexpected blast. This type of deafness may take from one to two years to reach an incapacitating point. Initially there is a severe high frequency loss, then a period of partial recovery, followed by regression toward an increasing profundity of deafness over a period of many months. Persistent exposure after the onset serves to aggravate the traumatic state. The traditional teachings in otology advance the idea that repeated exposure, such as that sustained by gunnery instructors, is most often represented in deafness from heavy firing. In our numerous series, however, there are only a few instances in which this was so. The diagnosis of "deafness from heavy fire" is reserved, incidentally, for those cases in which the evidence is incontrovertible.

In the analysis of chronic, progressive types of deafness we include those cases in which the tympanic membranes are normal and the losses are definitely of the conduction type, with negative Rinne tests and with evidence of gradually increasing profundity of loss. It is worth while to note that the hearing losses in this group seem to be far greater than in a comparable civilian group, the intensities in most instances going above 60 decibels, with audiograms presenting definite impairment of hearing for high frequency. This particular fact was revealing when we sought to find cases that would lend themselves to the fenestration operation.

In reference to the general category of nerve deafness, our cases run the gamut of etiologic factors, including childhood exanthems in which possible toxic factors were productive of deafness. Also included are a number of cases in which the history indicated impairment prior to induction; in these various injuries had been sustained, the trauma of skull producing deafness. Even preinduction meningitis has been noted in the histories of a few cases; these evidently escaped detection because of the dependence on conversational voice tests in preinduction physical examinations and because of inadequate testing procedures carried out at that time.

So far as auditory function in nerve involvements is concerned, some interesting observations have been made. A random sampling of 200 patients produced evidence that 56 per cent had been exposed to noise and blast, while 75 per cent suffered from severe tinnitus. Our patients commonly report severe headaches, recurring frequently, together with symptoms of vertigo. Without question, these data point to a relationship between nerve involvement in general and the symptoms of deafness produced by heavy fire, perhaps even to the central origin of deafness of this type. The relation of headache and tinnitus to normal communicative activities is obvious, and so far as civilian problems are

concerned, this whole topic needs much illumination in terms of industrial accident and compensation.

It is not amiss to comment on the fact that 4.4 per cent of our patients were inducted with chronic suppurative otitis media. Others were inducted whose membrana tensa was entirely absent; still others, with what appeared to be insignificant perforations in Shrapnell's area; and there were many with atrophic scars of healed perforations which failed to hold up under the conditions of service.

If, on the basis of our experience with cases of deafness existing prior to enlistment or induction, we were to make any positive recommendation regarding the disposal of persons with preservice hearing disabilities, it would be that no one be placed on active duty whose ears have been previously affected by inflammatory disease or whose tests—particularly the whispered voice—show the least diminution of hearing. Had these restrictions been operative in the past two and a half years, 40 per cent of our cases of bilateral deafness would have been eliminated, and there is no question that the lack of such restrictions contributed greatly to the numbers of those whose hearing became disturbed as a result of experiences in service.

Data from Questionnaires Returned by Former Patients.—A short time ago 1,172 questionnaires were mailed to patients who had completed the retraining course; to date 508 have been returned. On the basis of 490 of these replies, the following data are reported:

1. A total of 94 per cent are still using their aids. The reasons reported by the 6 per cent who are not using their aids include: batteries needed (5); repairs needed (4); no help to hearing (3); aid unnecessary because of regained hearing (3); aid causes headaches (2); ear mold causes soreness (2); aid cumbersome (2); aid too noisy (2); aid causes nervous strain (2); public disapproval of aid (1).

2. A change of hearing is suspected by 33 per cent.

3. Difficulty in obtaining batteries, presumably through the Veterans Administration, is experienced by 24 per cent.

4. Difficulty in obtaining satisfactory repair service has troubled 9 per cent.

5. A total of 23 per cent report that they have been refused a job or have been unsuccessful in holding a job because of their hearing loss.

6. To the question, "How much has speech reading helped you to understand conversation?" 52 per cent answered "greatly," 35 per cent "moderately" and 13 per cent "slightly."

7. The study of speech reading has been continued since discharge by 45 per cent; we assume this to be in the form of individual practice rather than formal instruction.

8. Suggestions for improving the program here included: (*a*) more speech reading; (*b*) more acquaintance with the workings of the Veterans Administration; (*c*) more practice with the hearing aid; (*d*) higher disability pension.

Procurement of Trained Personnel.—Obviously any program of the scope that is described here calls for refinements of judgment and technics that simply do not accrue from a casual interest in the problems of aural rehabilitation. Yet any realist is only too well aware that such organizations as ours in Philadelphia cannot immediately be set up for civilian service throughout the country, wherever they are needed. Let us assume that the financial support is available; from what source are sufficient trained specialists to be procured?

One group is readily available. Between the Army and the Navy Program of Aural Rehabilitation, at least 250 trained persons are ready to hand. Many of them came into the service organizations with rich backgrounds and high professional skill in working with deafened persons. Others have been specially trained within the services. In the navy two steps were taken. A training course in aural rehabilitation was instituted at Hunter College, New York, and for personnel in service a training program was instituted at the naval hospital in Philadelphia.

A brief description of the requirements and the content of the course at Hunter College will make clear the scope of this preparation. The minimum qualifications are a high school diploma and some experience in special fields of the work for the handicapped; desirable qualifications include college training with technical preparation in topics related to aural rehabilitation. The subject matter is divided into four categories: (1) the work being done for persons with auditory losses (a survey); (2) the anatomy, the physiology and the pathology of the ear and the vocal mechanism; the physics of sound; audiometry and the fitting of hearing aids; (3) the theory and the practice of teaching speech reading; the methods of training residual hearing, with the use of acoustic equipment; phonetics, and speech and voice problems of the deafened; (4) the psychosocial adjustment, with special emphasis on the psychology of the deaf and the hard of hearing. To this is added practice, which includes observation at otologic clinics and at public and private agencies for the deaf and the hard of hearing, supervised teaching of speech reading and auditory training, clinical work in the correction of speech and supervised use of all types of acoustic equipment.

There is no question that control of the qualifications of specialists in hearing clinics is an immediate and pressing problem. Nor is there any need to be satisfied with something less than thorough technical preparation. Many of these courses are offered at various educational centers; others can be instituted at selected colleges and universities.

Medicosocial Workers.—Medicosocial workers of the American Red Cross are attached to the program as trained consultants and render distinctive service. Their position is partly liaison, partly instructional and partly advisory. As liaison officers their work is twofold. They interpret the hospital situation and structure to the patient; they offer a bridge between his private affairs and his military status. As instructors, they help to indoctrinate the patient in hospital routine, help him adjust to the intensive training program, organize and supervise a variety of social and recreational experiences and teach him a diversity of arts and skills to occupy his time and broaden his interests. Special problems involving personal, familial and economic securities arise frequently, usually with attendant emotional instabilities; responsibility for handling these situations is shared by the medicosocial specialists. The medicosocial worker enjoys a unique position in the department. Because she is trained in every phase of the work and is in active touch with every element of the program, it is often through her initiative that problems of readjustment are solved.

The Use of Ear Defenders.—One aspect of the conservation of hearing to which naval specialists are addressing their attention is the efficicacy of various types of ear defenders in lessening acoustic trauma. Data are being gathered to enable us to examine the utility of various ear defenders with much more objectivity than the exigencies of the war have allowed. As yet, however, there remains the necessity to set up many more carefully controlled and more extensive experiments than have heretofore been reported.

Experimental evidence reported by the Medical Field Research Laboratory, Camp LeJeune, North Carolina, based on the exposure of a group of men to the fire of six 155 mm. guns, indicates that: 1. Almost any type of ear plug is effective in preventing significant hearing losses if the exposure is not too extensive. 2. In the situation tested there is little significant difference between cotton, NDRC V-51R and the modified V-51DK plugs; exceptional protection was indicated only for the defender made from acrylic resin. 3. Prolonged exposure to small arms fire while cotton plugs are used will result in significant hearing losses, with the greatest depression in acuity occurring at 4096 cycles per second. 4. All subjects, with or without ear defenders, suffered an immediate depression in hearing acuity, greater for the high frequencies; recovery was quicker and more complete among subjects using some sort of protection than among those who employed no protective device.

This problem of protection against concussive sound waves needs considerable illumination. Nor is it principally a military question, for the involvements in many types of heavy industry are considerable.

Situational Deafness.—Among various involvements of the auditory function, naval otologists recognize what may appropriately be termed

situational deafness. These are cases in which a patient reports a history of hearing loss, ordinarily associated with exposure to gunfire, blast or the noise of heavy machinery, and is supported by evidence from both functional and audiometric tests. The duration of the disability is relatively short, however, and occasionally, after a change in environment and the passing of time, the hearing acuity improves to a considerable extent.

Meantime, through fear of censure or the accusation of malingering, the patient continues to state that he does not hear. As he is processed from one facility to another he becomes more and more involved in his deception and the fact that numerous examinations further his cause; the only way out, he figures, is to maintain a now thoroughly trained response to bear out a nonexistent disability. The demonstration of bizarre discrepancies and a careful approach on the part of the examiner usually break down the barriers.

To date the instances of situational deafness presented at the center in Philadelphia have been few. Perhaps a good reason for this is that so many of our patients have suffered acoustic trauma for a long time before they reach us. With nerve deafness from heavy fire, a man has passed well through the cycle of hearing loss before he is admitted to the program. Later, after his training here, he may recover a little more hearing, but there is no evidence to show any miraculous recovery of full capacity after a serious nerve involvement. The contrary is frequently true.

The Navy recognizes, of course, that there are cases of malingering. We have had a few of these, usually among the lowest intelligence groups, and ordinarily picked up in the routine testing procedures. More frequently we have encountered patients who have tried to disguise their deficiency in order to remain in the service.

Situational deafness, however, is quite another matter, a legitimate corollary of exposure to blast and noise. Although the initial deafness may be only temporary, it is nonetheless profound; it is a true sensory experience, and the symptoms persevere long enough to invoke a definite pattern of response to the disability. With diminution of the symptoms, a man finds himself still responding to the behavioral pattern of deafness and is apt to become confused in the attempt to rationalize his recovery of hearing with the recent experience of profound deafness. This confusion leads him to continue to behave as though the symptoms were present, and a situation is set up which amounts to a *de facto* deception. This is a topic that will require more careful consideration in the future in terms of compensation involving aural disability.

Quantified Measurement of Hearing Acuity.—The suggestion has already been made in this discussion that audiometry as carried out in the naval program includes determining the hearing threshold for

discrete frequencies and also that for speech reception in a free field and that, although the one is quite as useful as the other, if the examiner were faced with a choice between the two, he might be closer to knowledge of a patient's practical hearing if he knew the level of speech reception. The precise correlation between these two measurements has not yet been worked out. In our procedures it is typically fairly close but not infrequently exhibits deviations both above and below a 1 : 1 ratio.

For example, it is not unusual for us to find that a man who has been deafened within a period of a year has a discrete frequency level (averaged for the conversation range) of 60 decibels of hearing loss but a speech reception level of 40 decibels of loss. The matter of correlation between the two tests is, then, apt to be variable; factors of age, duration of loss, tinnitus, condition of the tympanic membranes, intelligence and personality all enter into the picture.

By no means the least important item is the factor of speech memory. Whereas a patient may register a moderately severe loss so far as the measurement of hearing for pure tones is concerned, his lifetime habits of communication may come to his aid; his memory of speech may be such that he can understand with minimal aural cues. Another man with the same loss for discrete frequencies may be unable or unwilling to signify communicative intelligibility at such a level. Moreover, there is considerable evidence that the longer the duration of deafness the less acute is speech memory.

Correlations of our tests are now being prepared and will be published soon. Let nobody suppose, by the way, that this is an academic question. In the fitting of hearing aids the audiogram or threshold for discrete frequencies furnishes needed information; but if a disparity exists between that level and the loss for speech reception, the threshold of speech reception usually determines the amount of amplification necessary. One hopes that the means for the adequate measurement of speech reception will be available to every otologist in the near future. One assembly of equipment is sufficient to handle a considerable number of cases. We have as yet only one in Philadelphia and have processed over 1,600 patients in the past six months.

Hearing Aids.—Our intensive experience in Philadelphia has resulted in the development of certain standards and convictions relative to the performance of hearing aids. As yet there is no such thing as one hearing aid that is capable of all-around performance. We shall indeed be fortunate when the day comes that brings an all-duty hearing aid to the market. This is a feasible idea, by the way, that is even now in the laboratory stages.

Such an aid would embody the following features, which the specialists in our unit—technicians and instructors alike—have agreed on:

1. It would be a single transmitter (with as few varying models as possible) together with a variable tone control which actually governs the tonal complex reaching the ear.
2. It would incorporate adequate suppression of ambient noise beyond certain maximal sound pressures and thus lend itself to a variety of maximal tolerance levels.
3. It would present frequency amplification extending over a range from 250 to 400 cycles per second, and would contain a control to regulate amplification.
4. It would include a combination battery pack, sufficiently adjustable so that it might be packaged with the transmitter or worn as a separate unit.
5. It would include a standard receiver built to accommodate the maximal variables of the "perfect" transmitter.
6. It would probably utilize some kind of plastic-treated cord, which we have found to produce less of the disconcerting cord noise that is so often the cause for complaint. At present the plastic cords on the market are not as durable as they should be.

Evidence of improved design in recent years leads us to hope that such an ideal hearing aid will be available before long; when it is, some of the problems of aural rehabilitation will be automatically solved.

Fabrication of the Individual Ear Mold.—The importance of a properly fitted ear mold has already been referred to. It has been determined in our acoustic laboratory that in comparison with a properly constructed individual ear mold the "universal" ear pieces so often used may lessen the efficiency of a vacuum tube hearing aid by as much as 20 decibels. Nor can too much emphasis be placed on the care with which the "tailoring" of the ear mold is done. We believe, moreover, that the old technic of pouring an ear impression with a plaster mix, awkward, uncertain and finicky as it is, is thoroughly outmoded. We have developed a method of fabricating ear molds from a methylmethacrylate resin that is simple and expeditious; four trained technicians can make as many as thirty ear molds within a twenty-four hour period. All materials used in the naval laboratory are formulas of Samuel Cooper Plastics, Philadelphia.

We hold no special brief for the technic employed here. With minimal variations it is used in other centers, and the process is familiar to any dental technician. During the first months of our work in aural rehabilitation, we employed commercial sources in the city to take the

impressions and make the ear molds. This arrangement proved cumbersome, and we could not arrange delivery fast enough to meet our needs. Accordingly, we determined to set up our own prosthetics laboratory at the center, and since March 1945 our technicians have fabricated 1,600 ear molds.

Patients are assembled early each morning for the making of impressions of the selected ears. These are taken with a viscous plastic that is packed carefully into the external auditory meatus so as to cause no undue displacement of tissue. When the technician is satisfied that he has achieved a good impression—only ten minutes is required from the time the material is introduced into the ear—he numbers the semiplasticized material for identification and delivers it to the laboratory.

This impression is boxed in wax and a cast made by pouring a thin mix of artificial stone and plaster of paris. When this cast is ready, it is in turn filled with a preparation of the plastic material, packed in a dental flask and cured for four hours in a thermal bath at a temperature of 160 F. The flask is cooled, and then the plastic mold is dug out and ground to eliminate excess. The finishing process is begun by "skinning" the powder-like film from it with a dental burr; it is then brushed and polished, and with extreme care a canal is drilled to conduct sound waves. Finally, the mold is given a high luster with the use of a dry rag wheel and a high shine medium. It is delivered twenty-four hours after the making of the initial impression.

The patient wears his own ear mold during the evaluation and selection of hearing aids. Later, when his chosen aid is in daily use, the instructors in auditory training keep close track of the efficiency and the comfort of the ear mold in actual operation, and any necessary modifications to relieve pressure are made immediately.

TREATMENT AND TRAINING OF THE HARD OF HEARING AMONG CIVILIANS

Translation of Military Methods.—Let us now return to the second point of our original thesis: that the methods and the experience of those in charge of the military programs of aural rehabilitation can be translated and applied to correlative civilian problems and thus answer a great need both in the retraining of the hard of hearing and in the conservation of hearing. Certain aspects of the program outlined here, elements that are distinctly military, will not obtain in a civilian organization; other elements of a civilian program, such as the need to carry out rehabilitative measures without the controls of a military structure, will pose further questions that must be given careful consideration. By and large, however, there seems to be no apparent reason why the approach and the methodology developed in the service hearing centers

cannot be applied to civilian hearing clinics. The translation needs only boldness and the resolution of interested professional groups.

It is not argued here that these ideas are revolutionary. To date most of the intensive professional concern for the treatment and training of deafness has been turned to the congenitally deaf. Clinical and reeducational efforts on behalf of the hard of hearing remain undeveloped, even though they are carried on sporadically in various places throughout the country. Every specialist in hearing problems is of course cognizant of the efforts exerted by various leaders and groups —the prospectus of the American Academy, for instance, and the work of the Academy's committee on the conservation of hearing—to extend the treatment for aural disability and a program of conservation of hearing to a nation-wide structure. As yet, however, there is little evidence that procedures have gone beyond the general, administrative planning stages. We are concerned here with the establishment of hearing units to perform the necessary service. We believe that the naval principles and methods of aural rehabilitation described, based as they are on a wealth of practical experience in the field, provide an archetype for the clinical treatment and training of those with aural disability.

The necessary steps in the procedure of examination, the evaluation and selection of hearing aids, the retraining of residual hearing with the use of an aid and the development of skill in speech reading are clearcut. There are other ways of performing some of the tasks described here, but these have survived by reason of a successful demonstration, and this under conditions not always ideal. Perhaps if we think in terms of three types of establishments that may serve civilian and future veteran needs of aural rehabilitation, we shall cover the ground reasonably well.

The University Medical Clinic.—The first of these is a hearing clinic in a university center. Envisioned here is a complete rehabilitative organization, scaled much as is the Navy's center in Philadelphia, fully staffed and fully equipped to assume every responsibility relating to problems of hearing disability, speech disorders, conservation of hearing and training of speech habits. Such an organization would encompass three associated aspects of rehabilitative work: (1) the treatment and retraining of persons handicapped by defective speech and hearing; (2) education, including the training of professional personnel and the dissemination of public information in the field; (3) research on every aspect of disorders of speech and hearing and reeducation. It is assumed that some of the treatment and the training of the deafened in such a center would be conducted on a fee basis; for the most part, however, the clinic's work would be categorized as education services and would be so organized and financed. Moreover, aural rehabilitation offers an

excellent opportunity for a much needed and progressive type of philanthropy.

A university center, preferably one directly associated with an otolaryngologic department, seems to lend itself best to this design. A few such centers already exist; their number should be increased many fold. Facilities must be ample and personnel sufficiently specialized to produce good work and to conduct the rehabilitative process with thoroughness and dispatch. Such centers should assume leadership in training professional personnel and in disseminating to various state and community organizations the knowledge of methods and practices necessary to meet local problems and local needs. This knowledge must be drawn from the clinical experience of the university hearing center and extended through a responsible research program that is devoted to the problems in the field. The military is anxious and enthusiastic about continuing its productive relationship with universities interested in the field of hearing.

The Metropolitan Clinic.—A somewhat different type of clinic should be available in the metropolitan areas. Here, it is assumed, efforts are directed solely toward the rehabilitative task, with no special provisions for public-educational and research projects. A feasible organization may readily be developed in conjunction with major hospitals in the areas, the hearing clinics to be set up as special hospital services.

One assumes, too, that such a service must be self supported. For the sake of concreteness, let us examine a hypothetic state of affairs in a city with a population of 1,000,000 and a public school enrolment of 250,000. The usual statistical assumption is that from 2 to 5 per cent of the school population is hard of hearing; the middle ground of 3.5 per cent provides a potential for hearing rehabilitation of 8,750. The potential of adults would be considerably higher, say 11,250, at a conservative estimate. One is provided, then, with a potential 20,000 persons who would profit from treatment and training for hearing disability.

We envision a hospital-centered hearing clinic that is sufficiently well equipped to provide all necessary rehabilitative services. Approximately $15,000 would provide the necessary equipment. An annual patient load of about 1,000 would require a minimum clinical staff of 10, including 2 otologists and 8 nonmedical personnel. An annual budget of $40,000 will provide for such a staff and assure expert work. Allowing for reasonable expenditures for space, heat and power and the amortization of the cost of equipment, service in such a hearing clinic can be provided at an average cost of $45 per patient. This hypothetic city would need from three to five such clinics in order to furnish adequate service for the hard of hearing population.

This, it is believed, is an entirely reasonable state of affairs. Such a clinic would furnish all necessary services for diagnosis and treatment, for evaluating and fitting hearing aids, for training the patients' residual hearing and for teaching the fundamentals of speech reading. It might be recommended that patients continue to develop skills in speech reading by private arrangement, once the clinical service had supplied the groundwork. For all other rehabilitative services, the clinic would continue with follow-up consultations.

Aside from the usual amount of outpatient service, it is assumed that all retraining procedures would be conducted on a fee basis. Whether this might be a direct exchange between patient and clinic would depend on certain local considerations. Many cities are even now instituting programs for the hard of hearing through the public school systems; if excellent hearing clinics have been established by private agencies, arrangements may be made with the appropriate municipal departments to utilize the clinical services available. In some states such arrangements would of necessity be made or shared with the various state departments for the education of the handicapped. In any event it seems logical to believe that, once these services are available, enabling legislation, either state or local, can be arranged. The veteran problem is something else again. Plans for making rehabilitative services available to veterans are in a formative stage; we may assume that the services of some of these metropolitan and university clinics, as well as those of the military centers already established, would be employed for the veterans.

Leagues for the Hard of Hearing could have their efforts directed into appropriate channels to serve their communities on a higher scientific plane than they have heretofore. Their activities would thereby have a more universal impact on the problems of hearing loss. The leagues have long felt the need of being given a more definite role to play in aural rehabilitation, and now is the propitious time for them to assume this responsibility.

In summary, the transferral of the methods and the principals of aural rehabilitation developed recently by the Army and the Navy seems to furnish ready means to establish the structure necessary for the adequate treatment and training of the aurally disabled in metropolitan areas.

The District Clinic.—A third type of establishment would be necessary to provide essential aural rehabilitation in rural districts. One assumes that these hearing centers or mobile units would be substantially supported by state and district educational funds, although a certain volume of the patient population might receive service on a fee basis.

The precise nature of the rural speech and hearing clinic is somewhat more difficult to outline than that of any other type. Municipal and county hospitals would seem to be feasible sites, and certain minimal requirements of equipment and personnel are apparent. The services of an itinerant otologist might well be provided, as might those of a qualified acoustic consultant. Other basic rehabilitative services, such as competent audiometry, fundamental auditory training and instruction in speech reading, as well as educational and vocational consultation, should be at hand. These responsibilities could be assumed by college and university clinics for hard of hearing persons in some districts. In certain states, services for the adequate measurement of hearing acuity have already been extended to rural sections; it would probably be necessary, however, to supplement these broad studies of handicaps with thorough retests for those whose problems are specific.

Conservation of Hearing.—It has not been our intention to slight the conservation of hearing. On the contrary, it is assumed that all these proposed clinical centers for speech and hearing disabilities would do everything within their power to expedite general public educational and specific reeducational programs for the conservation of hearing. In the last analysis, all rehabilitative work with the hard of hearing is actually conservation of hearing, for psychosocial adjustment in terms of residual hearing is primarily conservation. In the broader view it is anticipated that the special work of the hearing clinics will be closely linked with educational and industrial efforts to lessen the incidence of hearing loss that is handicapping.

Summary.—Acquaintance with the military program of aural rehabilitation furnishes a rich background for thought in terms of the development of a program of treatment and training for the hard of hearing on a national scale. The Naval Program of Speech and Hearing Rehabilitation at the United States Naval Hospital, Philadelphia, dedicated to the psychosocial adjustment of the handicapped person and planned to achieve optimal retraining for communication within a four to eight week period, has thus far processed almost 3,000 aural casualties and furnishes ample data for transferring principles and methods to civilian hearing problems. This program has been described in considerable detail in the hope that individual workers and groups interested in aural rehabilitation will understand the Navy's practices and be stimulated to think in terms of applying similar procedures to the general civilian problems of aural disability and to the conservation of hearing. Special attention has been paid to the relations between medical and nonmedical services in the rehabilitative task. Certain findings and interpretations based on our experience are of particular interest. These have been discussed as separate topics and include such matters

as the range of hearing loss in the Navy, situational deafness, experiments with ear defenders, the fabrication of individual ear molds, the performance of hearing aids, the correlation between audiometric and speech reception tests and the procurement of trained personnel. Considering our experience with the Naval Program of Aural Rehabilitation, we have outlined a prospectus for three types of civilian hearing clinics, one for university centers, a second for metropolitan areas and a third for rural districts. It is believed that our principles could be translated into civilian needs within some such structure of speech and hearing clinics and go far toward solving a problem that has thus far received something less than sufficient attention.

WASHINGTON COUNTY (MARYLAND) PROGRAM FOR THE PREVENTION OF DEAFNESS IN CHILDREN

DONALD F. PROCTOR, M.D.
AND
W. R. WILLARD, M.D.
BALTIMORE

SURVEYS and studies have amply demonstrated that loss of hearing is an especially serious handicap to children. The "hard of hearing" child has difficulty (which neither he nor his associates understand) not only with his playmates and at home but especially at school. He may be thought careless, dull, headstrong or stubborn when he does not conform or fails or seems to refuse to follow directions. Depending on the degree of hearing loss, it is not unusual for a partially deafened child to be in school several years before the defect is recognized.

It was with these thoughts in mind, together with the more comprehensive idea of a full public health program for school children, that the health officer of Washington County (Maryland) determined to attempt to find and, if possible, cure remediable partial deafness in school children. Accordingly, in 1942 tentative plans were made for "an ear, nose and throat clinic" to be held in Hagerstown. The clinic actually went into operation in February 1943 and was known as the Washington County Clinic for the Prevention of Deafness in Children. During the first twenty months more than 600 patients were examined in the clinic. Although hearing improvement may be measured, there is no way of calculating how much hearing impairment may have been prevented.

Aural infection and hearing impairment are relatively common conditions, particularly among children of school age.[1] The mental effect of hearing impairment as revealed in school work and in the pursuit of a gainful occupation in adult life needs no elaboration. Depending on the type of tests used, between 3 and 30 per cent of school children have been found in various surveys to have some hearing impairment. On the average about 5 per cent of school children have sufficient hearing impairment to be impeded to some degree in their understanding of speech. The percentage of those whose hearing defect is due to a congenital lesion (in our series to date less than 1 per cent),

1. Willard, W. R.: A School Medical Program for the Duration, South. M. J. **36**:503-507 (July) 1943.

to otosclerosis or to remediable conditions, such as otitic infection or nasal disease associated with damage of the middle ear has not been adequately determined. It is certain, however, that a large proportion belong to the last category, and it is possible that 80 or 90 per cent of school children with impaired hearing can be helped by proper therapy.

Hearing impairment and aural infection are commonly contraindications to employment in both the Armed Services and industry. The data thus far published about the examinations of draftees indicate that at least 5 per cent of all deferments for physical reasons were for otologic conditions.[2] Efforts to combat infection of the ear and deafness in the past have had discouragingly poor results. Recent developments in chemotherapy and improved understanding of the causes of deafness associated with disease of the nose and throat and its proper care (particularly the use of irradiation therapy to control lymphoid tissue interfering with the function of the eustachian tubes) [3] have led to the belief that concentrated effort can materially reduce and perhaps largely eliminate deafness resulting from such disease.

DEVELOPMENT OF THE PROGRAM

Early in 1942 the health officer of the Washington County Health Department, serving 72,000 people, became interested in the problem of the prevention of deafness. It was thought that the facilities and the resources of the department and the county could profitably be used for that purpose. Among the available resources was a modern, well equipped general hospital and five physicians limiting their practice to diseases of the ear, the nose and the throat.

The health officer conferred with (1) the otolaryngologic staff of the Johns Hopkins Hospital, (2) Dr. Curtis Burnam (who developed the radon applicator for treating adenoid tissue), (3) representatives of the Maryland League for the Hard of Hearing, (4) representatives from the State Department of Health, (5) the United States Children's Bureau, (6) state and county departments of education, (7) the county medical society and with other interested persons and agencies. Even-

2. Rowntree, L. G.; McGill, K. H., and Folk, O. H.: Health of Selective Service Registrants, J. A. M. A. **118**:1223-1227 (April 4) 1942.

3. Crowe, S. J., and Guild, S. R.: Impaired Hearing for High Tones, Acta oto-laryng. **26**:138-143, 1938; Recognition, Treatment, and Prevention of Hearing Impairment in Children, Laryngoscope **50**:658-662 (July) 1940. Burnam, C. F.: Irradiation Treatment of Hyperplastic Lymphoid Tissue, ibid. **50**:663-670 (July) 1940. Crowe, S. J., and others: Impaired Hearing in School Children, ibid. **52**:790-804 (Oct.) 1942. Crowe, S. J., and Burnam, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

tually an advisory committee was established in which these various interests were represented.

It was thought desirable that the program should provide a fair test of the maximum possible benefit from the most modern methods of treatment, that it should not arouse antagonism and that any successful methods developed should be of use in such programs elsewhere. Plans were made, therefore, to operate as a part of the program a clinic offering the best available methods of diagnosis and therapy with all needed facilities and instruments. The services of an otolaryngologist were obtained for two all-day clinics a month, and arrangements were made for him to cooperate in the planning of the entire program. Careful plans were made to limit the clinic's activities so as not to compete with local medical practice.

The United States Children's Bureau agreed to provide financial support for the program, the funds for which were to be administered by the State Department of Health through its services for crippled children. It was decided that each patient who was able to pay should be charged a fee of $5 for radon treatments, but no person was to be refused treatment because he could not pay. The fees would go to the support of the program. As the program developed, the following expenditures were found chargeable to the clinic: outlay for instruments and equipment; clinician's fee; technician's salary; drugs and supplies; rental of radon; provision of hearing aids, and payment of surgeons, anesthetists and the hospital for surgical treatment of the medically indigent. Chargeable also was the proportion of time devoted by the public health nurses and a clerk. Preliminary arrangements occupied almost ten months, and in February 1943 the clinic was ready for operation.

OPERATION OF THE PROGRAM

There have been no major changes in the functions of the clinic during its period of operation. Its activities may be divided into the following groups:

Case Finding.—It was planned that the clinic would accept patients referred by private physicians for consultation, particularly if a hearing defect was suspected or a condition was present which might lead to a hearing defect. It was also planned to examine children referred by teachers, nurses, welfare agencies and other authorities, after proper clearance through the family physician. It was further planned to institute screening of school children by means of routine hearing tests as a further source of potential patients. Arrangements were made with the Maryland School for the Deaf for an initial screening of Washington County school children, a service which that institution had been offering

to the counties of Maryland for several years. This screening, carried out promptly after the program started, located about 250 school children with apparent hearing defects, and 91 of the children with the more serious defects were seen in clinic during the first year. It was planned, however, that the routine screening for hearing defects among Washington County school children would become, after the initial survey, the responsibility of the technician in the program, who would test at least two grades each year throughout the county.

The various case finding methods have been most successful. In fact, the clinic has more requests for appointments than can be granted. In case finding, from the outset patients with aural infections, whether chronic or acute, were considered by far the most urgently in need of the service of the clinic. Next to these came patients with a history suggestive of hearing impairment. Speech difficulties and otherwise unexplained behavior problems in the schools were considered as possible clues to hearing impairment. Although this program was primarily for the purpose of preventing the development of deafness in children, it was felt that, at least at first, such conditions as these must be considered more urgent and that only if the time was available could children suffering from disease of the nose or the throat only potentially predisposing to disease of the ear receive the clinic's attention.

Medical Examinations.—The examination consists of an electric otoscope examination of the ear, palpation of the cervical glands, inspection of the teeth, the tonsils and the pharynx (the larynx when indicated), nasal speculum and electric nasopharyngoscopic examination, and transillumination of the sinuses. Cultures and roentgenograms have been made when indicated. The absolute necessity of a nasopharyngoscopic examination cannot be overestimated, and obviously the clinician must be trained so that he can make this examination. It should also be emphasized that the nasopharyngoscope can be used successfully with school children *of all ages,* and also with most preschool children, without difficulty.

On the average, 4 new patients can be examined in an hour, and about 10 return patients are rechecked in that time. The clinic begins at 9 a.m. and is usually over by 4:30 or 5 p.m.; but on occasion evening appointments are scheduled, if necessary, in order to see all patients. Clinics are held two or three days a month.

In taking histories the following points have been given particular attention: history of deafness or allergy in the family; infections of the ear or hearing difficulty; headaches; recurrent sore throat; unusually severe colds; nasal obstruction. Parents are urged to attend the clinic with their children so that adequate histories may be obtained. The history is recorded by the secretary as the physician asks the questions. She also records the results of the examination except those of the

hearing tests (recorded by the technician) and the diagrammatic representation of the nasopharynx and the ear drums (chart 1). The last items are recorded by the physician as: (1) anterior-posterior cross section of the nasopharynx; (2) view of the eustachian orifices seen medially; (3) profile diagram of the ear drum. Such diagrams have proved easy to make and easy to interpret.

Clerical Work and Records.—The history, the data from the physical examination and the notes on treatment and progress are all kept by

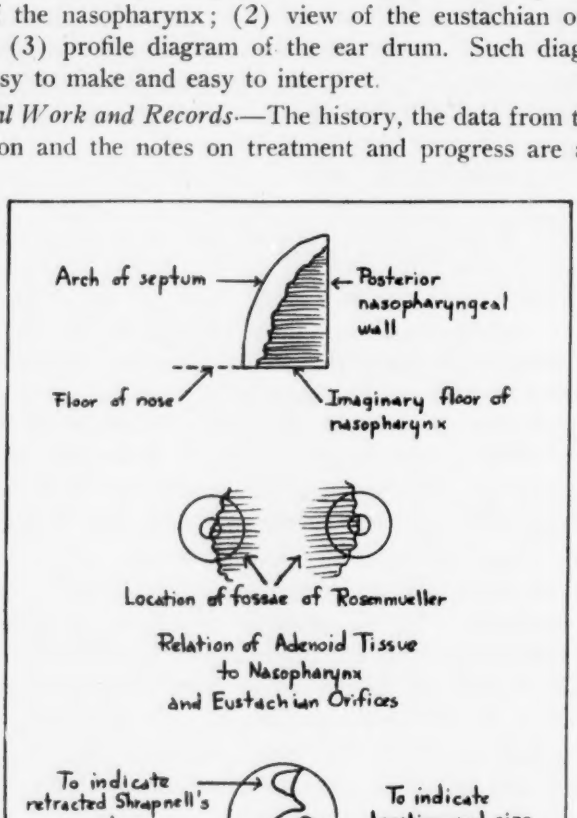
Arch of septum
Posterior nasopharyngeal wall
Floor of nose
Imaginary floor of nasopharynx
Location of fossae of Rosenmueller
Relation of Adenoid Tissue to Nasopharynx and Eustachian Orifices
To indicate retracted Shrapnell's membrane
To indicate location and size of perforation
Appearance of Ear Drum

Chart 1.—Clinic physician's diagram of the nasopharynx and the ear drum of a patient.

the secretary on a simple mimeographed record form (chart 2). Most of the information is on the printed side of the form and the back is used for additional notes, correspondence and other details. A card for each patient is calendar filed under the date of his next appointment, and notices are prepared from this file. A report of the clinic findings and recommendations is sent promptly to the referring physician or to the family physician if the patient has not been referred. This work requires about one third of the full time of a medical stenographer.

NAME Keller, Dolores Age 10 Sex F Col. W Parents' Names Mother Isabel Hoover Keller #100

* Address 324 Jefferson St. Referred by: Miss Annan - Trans. Miss Hull

Tel. # School Bury. Grade 4 Physician

✓ Not done in S.P. room

Right			Air. Cond. ✓	Thresholds S. P. R.	Decibels	2-A Aud.			Left
4/11/44	2/17/44	8/7/43	6/5/43	Date		6/5/43	8/7/43	3/12/44	4/11/44
10	25		30	64		30		25	15
10	25		25	128		30		30	10
15	35		40	256		45		35	15
20	40		50	512		55		40	20
15	45		50	1024		50		40	15
15/5	40		45	2048		55		30	10
10	40		40	4096		55		40	15
20	40		35	8192		35		35	15
30	40		40	11700		35		35	20
3ft.	3ft.	2ft.		Spoken Voice			2ft	3ft.	3ft.
2ft.	1ft.	1ft.		Whispered Voice			1ft.	1ft.	2ft.
				Weber		✓			
BC 25 AC 60	BC 30 AC 35		BC 30 AC 45	Rinne		BC 30 AC 35		BC 30 AC 40	BC 35 AC 50
4/22/44	2/17/44	8/7/43	6/5/43	Date		6/5/43	8/7/43	2/12/44	4/22/44
				DRUMS					
				ADENOIDS					
				Eust. Orifices					
Radon Treatments - Date			6/17/43			6/7/43	2/12/44	4/22/44	
Millicuries			123			80	114	113	
Minutes each side			2.25			3.75	2.9	3	
Gram minute equiv.			.28			.30	.33	.33	

History: Ear Otitis Media foll. T and A Earaches

Nasal Symptoms: None

Familial Deafness None Allergy None

Headaches None Sore Throat None

T & A age 7 Other

School referral - Impaired hearing on screen test - Mrs. Elliott

EAR Date 6/5/43

Thickening	None			
Scarring	None			
Culture M.M. Mid. Ear				

NOSE & NASOPHARYNX

Br. Sp. & Sep.	Adeq			
Discharge Culture Polyps	None			
Trans. Ant. Trans. Frn.	Not done			

MOUTH & OROPHARYNX

Teeth	Need attention			
Tonsils	Cleanly removed			
Base Tongue Phary. Walls	Normal			
Neck Glands	Not enlarged			

* Mother @ 324 Jefferson St.
Child c Mr. and Mrs. Mahoney either in Piney Pt., Md. St. Mary's Co. or 918 - 8th St., Washington, D.C.
Moved back to Hagerstown, March 1944

Chart 2.—Record of a patient kept by the secretary of the clinic physician. *T & A* signifies tonsillectomy and adenoidectomy; *S.P.R.*, soundproof room; *Br. Sp. & Sep.*, breathing space and septum.

Hearing Tests.—When the program was organized, a technician was selected for testing hearing and her training begun. She spent several weeks studying at the Johns Hopkins Otological Laboratory and under the consultant otolaryngologist, learned the technic of various hearing tests and something about their interpretation. During the first two months all tests were checked by the physician of the clinic, and even now an occasional test is checked. The precaution of checking the hearing tests occasionally provides a constant stimulus to accuracy, even though the technician be quite capable, experienced and conscientious. No soundproof room has been provided, although one is planned for the near future. Whenever tests are done under unusually quiet or under especially noisy conditions, this is noted on the record, and the fact that tests were not made in a soundproof room is always considered in interpreting them.

Three types of testing are done: 1. Young children (usually under 5 years of age) or uncooperative or very deaf children are tested with the 512 tuning fork and with the voice, an electric masker being used to exclude the ear not being tested. 2. Whenever the patient's ability to cooperate permits, a pure tone audiometric test is made in addition, and the thresholds for the frequencies from 64 to 11,700 cycles are determined with a Western Electric 2A Audiometer. 3. In the screening carried out at the schools by the Health Department's technician, all third and sixth grade school children are asked briefly about aural infections or hearing difficulties, and the ability to hear the spoken and the whispered voice (with the opposite ear masked) is carefully determined. In some of the work audiometric tests for thresholds at 512 and 8,192 cycles have been added to the screening test, and it is planned to use them routinely in the future. All children suspected of having some hearing deficiency as a result of this test or giving a history of aural infections are given a complete hearing test and sent to the clinic for examination. At present a little over one half of the technician's time is devoted to this program.

Treatment in the Clinic.—Simple local treatment, prescriptions for medication, cleaning of infected ears and other tasks of this nature have all been handled at the clinic.

Whenever elimination of the nasopharyngeal lymphoid tissue has seemed desirable, irradiation therapy has been given at the clinic. If there is a large mass of adenoid tissue, most satisfactory results are obtained by surgical removal combined with irradiation. For restoration of the function of the eustachian tubes, treatment is continued until the eustachian orifices are completely clear of lymphoid tissue; but in patients suffering from recurring colds, asthma and other conditions treatment should be continued until the entire nasopharynx is clean and covered with mucous membrane similar to that on the nasal septum.

Treatment Outside the Clinic.—For treatment locally available, including surgical, patients are referred to local physicians and surgeons. Patients continue attendance at the clinic after medical or surgical treatment. This is true of both private and free patients, and it is considered most important that this attendance be continued. Advice as to treatment is not enough; proper care must be carried out.

For surgical work recommended by the clinic to be done on patients from families certified as in financial need by the County Welfare Department, physicians, surgeons and the hospital are compensated at a schedule of fees established in accordance with minimum prevailing rates. Tonsillectomy and adenoidectomy are the most usual surgical procedures, and during the first year 33 tonsillectomies and adenoidectomies were paid for in this way.

When a hearing loss is found not to be amenable to treatment, the clinician advises about obtaining hearing aids and special education. When necessary, funds are provided for this purpose. The cooperation of the Baltimore League for the Hard of Hearing has been of great assistance in this respect.

Nursing.—A nurse assists at each session of the clinic and all the public health nurses assist in case finding, follow-up work and procuring cooperation between patient, clinic, private physician, welfare department, school and any other interested agency or person. The follow-up work of the nurses has proved an essential part of the program, and this applies not only to their service with so-called indigent but also to that with private patients. In the Washington County program remarkably conscientious work has been done by the nurses, and it is largely due to this work that satisfactory cooperation of the patient has been the rule, and refusal to cooperate the exception. The credit due to this particular staff of nurses cannot be overstressed.

REMAINING PROBLEMS

To date the difficulties encountered have been comparatively few. Certainly the program has been well accepted by the practicing physicians and the community. Appointments are not often broken, and so far only about 2 per cent of patients are found to be totally uncooperative. About 5 per cent of the patients sent to the clinic had no hearing impairment. Surgical work performed by the local otolaryngologists on clinic recommendation has been, in most instances, satisfactorily performed.

However, there are problems, as yet unsolved or incompletely solved. As previously mentioned, the demand for appointments exceeds all expectations and ability to grant them. To insure that the clinic serves the purpose for which it was organized and that it renders

the maximum service to the community, various steps have been required to regulate appointments. These steps have been taken in this order:

First the number of adults accepted has been so sharply reduced as to be almost negligible except for doctors, nurses and certain other patients in that category who can hardly be refused.

Second, the number of new patients referred by private physicians has been reduced. During the early months of the clinic two thirds of the patients were referred by physicians in private practice. Now the number is nearer one third and, correspondingly, more patients referred by school and health departments can be seen. This has been thought wise because, as a rule, patients referred by private physicians have had less serious otolaryngologic conditions than the other patients of the clinic; however, it has been difficult to refuse physicians who obviously want and need for their patients the service the clinic is prepared to give.

Third, a system of "priorities" (as previously mentioned) has been introduced to insure that patients urgently in need of attention will not have to wait an unreasonable length of time for appointments.

It is believed that in time the waiting list of children with neglected disease of the ear, the nose or the throat will be greatly reduced and that it will be possible after a few years to keep up with current demands.

Only partial success has been achieved thus far in securing for all patients the necessary surgical treatment, particularly tonsillectomy and adenoidectomy. Thus, of the tonsillectomies and adenoidectomies advised by the clinician, a little over half are known to have been done. However, a high percentage of those patients classified as urgently in need of tonsillectomy and adenoidectomy have received this surgical treatment.

A real problem exists, however, in securing necessary surgical work, and it has been aggravated by loss to the Armed Services of some of the local otolaryngologists. The problem is most acute, as might be expected, among patients in the borderline economic group, that is, patients whom the Welfare Department does not certify but who are unable to pay for the work.

It has been suggested that the employment of a trained medical social worker would help solve this problem, especially if the decision as to eligibility for financial assistance were put in her hands and removed from the Welfare Department, where comparatively inflexible criteria are used. It is believed that the medical social worker could also help families to solve problems other than the financial one which interfere with the securing of adequate treatment. She could further determine which patients at the clinic should be treated with free radium, which for a fee, and how large a fee. It is planned that a medi-

cal social worker will be employed when one can be obtained to do work for this and other medical care programs in the Health Department.

The record form has been revised twice but is still not completely satisfactory for statistical and analytic purpose. This fault will eventually be amended.

ACCOMPLISHMENTS

This is not intended to be primarily a report of results, as it is still much too early to expect many benefits or to interpret properly either successes or failures. The chief purpose of this report is simply

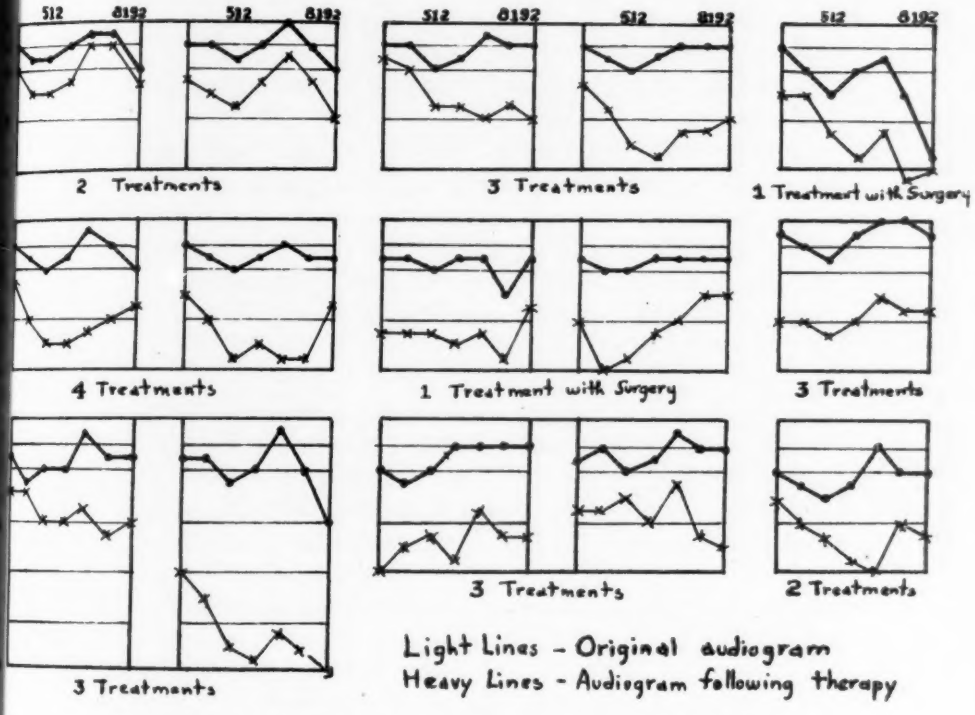

Chart 3.—Typical results of treatment instituted under the program.

to present the mechanism of a clinic in actual operation. However, the following figures will give some idea of the quantity of work which may be accomplished with such an organization.

As of the date of this report (July 1945), there have been approximately 40 clinic sessions, at which over 700 new patients (48 per cent females and 52 per cent males) have been seen in over 1,500 clinic visits. Thirteen per cent of the patients are over 20 years of age (half of these are no longer on the active list). Fourteen per cent of the patients are under 5 years of age, 36 per cent between the ages of 5 and 10, 32 per cent between 11 and 15, and 5 per cent between 16 and 20.

About 100 patients have suffered from chronic or recurrent aural infections, and over 250 patients have shown definite hearing losses, about 200 of these involving the conversational tones.

Of the patients suffering from unusually severe recurrent infection of the upper respiratory tract and treated by irradiation, 90 per cent have shown a decided decrease in frequency and severity of infection. Of the patients with chronically discharging ears, treated by irradiation and simple local therapy (no mastoidectomy), 58 per cent now have dry and apparently healed ears (only a few with intact drums). Of the patients suffering from frequently recurring acute infection of the ear and treated by irradiation, 71 per cent have had no further infection of the ear (followed for several months through the past winter). Of the patients subject to bronchial asthma and treated by irradiation, 39 per cent have had almost complete relief, and of those who have not yet been relieved, none has yet received adequate irradiation. One hundred and eighty ears with hearing impairment have returned to normal, and 90 ears with hearing impairment have improved. One hundred and forty ears with hearing impairment have shown no essential change as yet, and 25 ears have shown some increase of impairment (chart 3).

Typical encouraging results are shown in chart 3.

CONCLUSION

Within five or six years we hope that a high percentage of all children in Washington County suffering from early impairment of hearing, aural infection or disease of the nose and throat predisposing to such difficulties will have been seen at the clinic at the early ages when therapy is relatively simple and extremely effective. Within a generation or two the percentage of adults suffering from impairment of hearing or severe chronic disease of ear, nose or throat will be materially reduced if such a program proves successful.

In conclusion it should be stressed that in organizing any such clinic anywhere the following points are of the utmost importance:

1. An adequate, interested and cooperative health department is an absolute necessity for such a program.

2. The clinic should be equipped with all necessary instruments, including an electric otoscope and nasopharyngoscope, a reliable audiometer and an electric masker. Radium or radon must be available. Unless these conditions are met, little in the way of proper diagnosis or effective therapy can be hoped for.

3. No amount of planning can make up for the lack of a health officer and an otolaryngologist who are capable of meeting the specific problems peculiar to the community involved.

6 East Eager Street.

IRRADIATION FOR THE ELIMINATION OF NASOPHARYNGEAL LYMPHOID TISSUE

DONALD F. PROCTOR, M.D.
BALTIMORE

INFECTION in the nasopharyngeal lymphoid tissue commonly plays a uniquely important role in the causation of the common cold, deafness due to malfunctioning eustachian tubes, recurring or acute infection of the ear, bronchial asthma (especially in children) and chronic bronchitis. This is partly due to the anatomic location of such tissue, but a great deal about its etiologic role is as yet only poorly understood.

In many patients irradiation of nasopharyngeal lymphoid tissue is essential for its adequate elimination. Surgical removal of adenoids is at best not perfect, and even if all the nasopharyngeal lymphoid tissue could be removed, that within the orifice of the eustachian tube would not be affected. Lymphoid tissue is an integral part of the mucous membrane in this region and is bound to recur as the mucous membrane grows back. Lymphoid tissue is so sensitive to irradiation that minute dosages will cause its regression and eventual disappearance without damage to surrounding tissues.

Practically all spontaneous radioactive substances belong either to the uranium or the thorium group of elements. Radium, by far the most used and best known of the elements, is a member of the uranium family which starts with uranium I and ends with lead. The uranium series consists of:

1. Uranium I, atomic weight 238, which by spontaneous transformation decreases to one half in 4,670,000,000 years.
2. Uranium II, atomic weight 234, half value in 2,000,000 years.
3. Ionium, atomic weight 230, half value in 69,000 years.
4. Radium, atomic weight 226, half value in 1,600 years.
5. Radon, atomic weight 222, half value in 3.85 days.
6. Radium A, atomic weight 218, half value in 3 minutes.
7. Radium B and radium C, atomic weight 214, half value in 26.8 minutes and 19.7 minutes, respectively.
8. Radium D and radium E, with half value in 25 years and 5 days, respectively, are isotopes of polonium.
9. Radium F, or polonium, atomic weight 210, half value in 136.3 days, and finally lead, atomic weight 206.

Omitting from consideration the predecessors of radium and the descendants of radium B and radium C, there remain only radium, radon and radium B and radium C of importance in the nasal applicator. The effectual beta and gamma rays from a radium-containing tube are from radium B and radium C. Beta and gamma rays invariably are given off together.

The active part of the applicator is a Monel metal tubular chamber 15 mm. in length, with an inside diameter of 1.5 mm., a wall thickness of 0.3 mm. and an outside diameter of 2.1 mm. The chamber contains 50 mg. of radium sulfate. The absorptive capacity of a filter is proportional to its density. To absorb all beta rays only 0.5 mm. of platinum, gold or tungsten is required, 1 mm. of lead or silver, 2 mm. of brass or monel metal and 10 mm. of aluminum. Even when all primary beta radiation is absorbed, there are secondary beta and gamma radiation from the filter, which is most penetrating from metals of high density. These secondary radiations are not large from monel metal, and for applicator treatment of the nasopharynx a secondary screen of lighter material is not necessary.

Practically all the beta rays emitted by this applicator are absorbed by the tissues of the nasopharynx. About 75 per cent of the beta rays are absorbed in the first 3 mm. of tissue, but 6 to 7 per cent of the hard beta rays are still detectable after passing through 20 mm. of tissue. In contrast, less than 10 per cent of the gamma rays are absorbed. The effects aimed at and obtained by radiation therapy are entirely dependent on ray absorption, and are roughly proportional to the amount of absorption. Rays that pass through the body and are not absorbed have no therapeutic effect.

The therapeutic effect of a single dose may be modified greatly by the reparative powers of the tissues. A unit dose given at one sitting produces more effect than the same total dose given in daily fractions in a period of a week. After moderate irradiation the nasopharyngeal lymphoid tissues recover in a period that varies from three to four weeks. Therefore the interval between treatments has been fixed at twenty-five days. Edema is a constant change following irradiation. Therefore, it is advisable to omit treatment when the patient has acute nasopharyngitis and in subacute nasopharyngeal infection to give half the usual dose at the first sitting and the other half five days later. Twenty-five days later the second treatment (six minutes and forty seconds on each side of the nasopharynx) is given, and if nasopharyngoscopic examination indicates further treatments are necessary, they must be spaced at intervals of twenty-five days. As a rule three treatments are adequate, but if the tissue is refractory, four or five may be necessary. If the adenoids are large, a combination of surgical removal and

one immediate irradiation of each side of the nasopharynx for six minutes and forty seconds is more effective than three or four radiation treatments alone.

Cells vary in sensitivity; next to sex cells, lymphoid tissue is the most sensitive in the body. Therefore, it is possible to treat adenoid tissue with a dosage far below the level that would injure mucous membrane, bone, cartilage, ductless glands or the central nervous system, including the eye. Hyperplastic tissues are more easily injured than quiescent tissues. Pathogenic bacteria, which cannot be destroyed in cultures, often disappear following irradiation of tissues in which they are located.

The size of the nasopharynx and the state of the lymphoid tissue vary, and the effects of treatment are not the same in every case. One is treating not deafness or aero-otitis but conditions in the nasopharynx that favor the development of these maladies. The aim of the treatment is to remove mechanical obstructions in the eustachian tubes and the chronic infection in adjacent lymphoid tissue which causes hyperplasia, excessive secretion of mucus and edema of the mucous membrane.

The dosage that has been found most effective with this applicator is "1 Gm. twenty seconds" on each side of the nasopharynx—since this applicator contains only 50 mg., or one twentieth of a gram, the dosage is twenty times as long—four hundred seconds, or six minutes and forty seconds, on each side.

Should the applicator contain an appreciably smaller or greater amount of radium (1 to 2 mg. either way will make no difference), the time it is left in the nasopharynx will have to be increased or decreased to approximately the desired dosage of "1 Gm. twenty seconds." For example, if the applicator contains only 40 mg. of radium, the strength will be only one twenty-fifth of a gram and the time should be increased to five hundred seconds, or eight minutes and twenty seconds.

The nasal applicator must be treated as a delicate instrument. The beta and the gamma rays given off are constant so long as the chamber containing the radium salt is hermetically sealed. A microscopic fracture, not jeopardizing the radium, may cause loss of radon and reduction in gamma and beta output. The applicator should be tested for a radon leak once a week with an inexpensive alpha ray electroscope. Place the applicator in the ionization chamber of the electroscope, leave for a few minutes and then remove. When no radon is escaping, the electroscope can be charged as usual. When radon is escaping, the electroscope becomes "contaminated" and will not function for hours. A leaking applicator should be returned to the radium company for repairs.

TECHNIC OF TREATMENT WITH THE RADIUM APPLICATOR

1. The applicator should be left in the lead cylinder until one is ready to insert it into the patient's nasopharynx. A lining tube of glass may be fitted into the hole in the lead cylinder and kept filled with 95 per cent alcohol, which is a convenient means of sterilizing the applicator. Heat must never be used.

2. The patient should lie comfortably on a couch during the treatment.

3. The alarm time clock should be used to control the duration of the treatment.

4. Remove the applicator from the lead cylinder, wash off the alcohol with water, dip into a lubricant (one that is water soluble so that it may be quickly washed off at the conclusion of the treatment) and insert into the nasopharynx.

5. Exact placing of the applicator is most important. The nasopharynx varies in size. The middle (not the end) of the radium-containing chamber should be in direct contact with the tissue in the fossa of Rosenmüller or the orifice of the eustachian tube. The exact location and the depth from the external nares to the area to be treated can be determined only by a preliminary examination with the nasopharyngoscope in one side of the nose and a calibrated rod or applicator in the opposite side. Never bend the applicator in order to put it in the eustachian tube, since this may lead to leakage of the radon. When it is in place, fix it with a suitable clamp or adhesive.

6. The most important protection for personnel using the treatment is distance. The intensity of the radiation varies inversely with the square of the distance. At a distance of 1 foot the square is $1 \times 1 = 1$, which is called unity. At 10 feet it is $10 \times 10 = 100$, and the intensity is 1/100 of that at 1 foot. At 20 feet, it is $20 \times 20 = 400$, and the intensity is reduced to 1/400 of that at 1 foot. During the treatment, all personnel who are constantly handling the applicator should be at a distance of 25 or 30 feet (7.5 or 9 meters).

7. At the conclusion of the treatment, the mucus adhering to the applicator should be removed with a brush fixed to the table or the sink and the applicator quickly replaced in the lead cylinder. The radium-carrying chamber at the end of the applicator must never be touched. If the applicator is repeatedly cleaned by wiping it with gauze held in the hand, radiation changes will soon become evident in the skin and the nails.

8. The nasopharynx is reexamined at the end of three to four weeks and treatment repeated at this interval and subsequent intervals until satisfactory regression has occurred.

If the precautions outlined are followed, there is no danger to the hands or the fingers. The applicator should always be held at the end of the handle and away from the body. The placing of the applicator should be done rapidly, and when treatment is completed the applicator should go back into the cylinder immediately.

Over-radiation of the entire body daily for months will produce a drop in the white blood cell count. This is mainly in the lymphoid cells. One tenth of an r of gamma radiation per diem is without cumulative effect. Actually, in treating 50 patients a day and every day, if the precautions indicated are followed, the body would not be exposed to anything like this safe dose—probably not more than 1/20 r per diem. There may be variations in tolerance, and it should be a routine practice to make a complete cell count of the blood in every doctor and nurse employed in these treatments. Counts should be made every two months. Persons who at the onset show a low white cell count must be excluded from the work.

RESULTS OF RADIATION THERAPY

An applicator containing 50 mg. of radium salt within a filter of 0.3 mm. of monel metal has been used in the work at the Hagerstown Clinic for the Prevention of Deafness in Children for the past eight months. This type of radiation therapy has been in use at the clinic for more than two years, with 323 patients being given to date nine hundred and eighty-one treatments. Many of the patients still have treatments to undergo, but 100 patients have had complete eradication of nasopharyngeal lymphoid tissue, and 144 patients have had satisfactory regression of such tissue with clearing of the eustachian orifices. Only 10 patients have remained completely unimproved, and only 2 of these have received more than two treatments (chart 1). Since radiation therapy produces results only over relatively long periods (at least two months and often considerably longer) it is difficult to evaluate results of therapy in a clinic which has been in existence only a little over two years. So far, however, my associates and I are convinced that the following results may be attributed to the radium treatments:

Nineteen patients with recurrent otitis media have remained well since treatment.

Twenty-six patients with chronic otitis media have now had dry ears over a period of at least many months.

Eight children with bronchial asthma have ceased to suffer from attacks, and 2 other children have been greatly improved.

Forty-three patients with unusually severe recurrent infection of the upper respiratory tract remained relatively free from this affliction for at least the past winter.

Nineteen ears with severe impairment of hearing have returned to normal and 268 ears have shown definite improvement of hearing (chart 1).

It is necessary to speak of ears instead of patients in considering improvement of hearing because of frequent discrepancies between ears of an individual patient. The importance to the patient of improvement of the hearing of one ear is almost equal to improvement of the hearing of both ears.

Approximately half of our patients with improvement of hearing have shown this improvement in both ears. An occasional patient has had improvement of the hearing in one ear while the hearing in the other ear has continued to grow worse. In 17 ears in the group under study the hearing has grown worse under treatment.

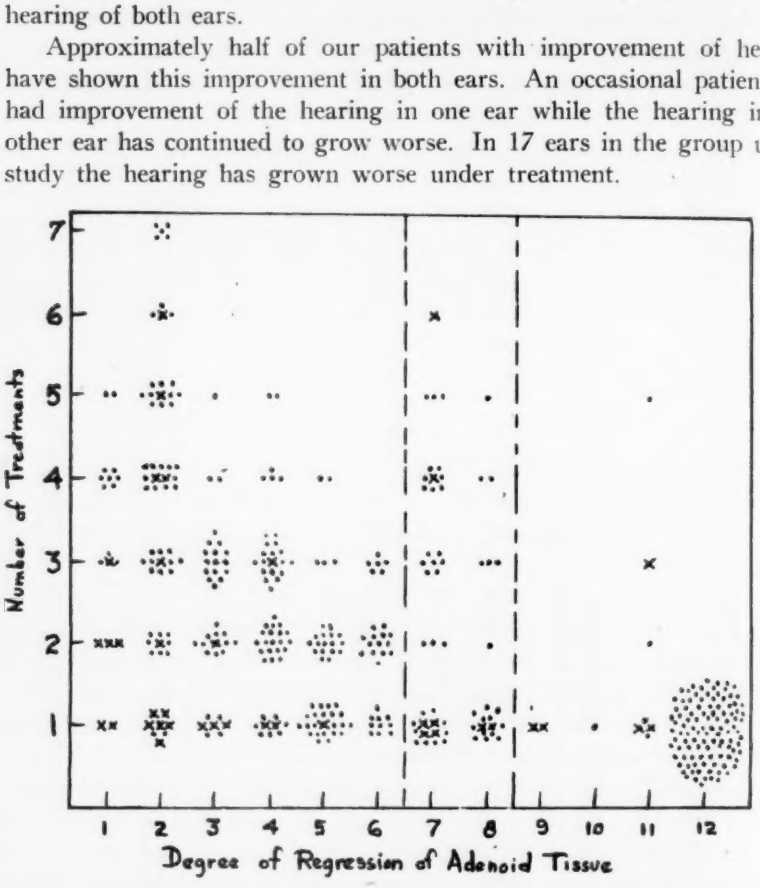

Chart 1.—Results of treatment aimed at removal or destruction of nasolaryngeal lymphoid tissue obstructing the orifices of eustachian tubes. Irradiation with surgical treatment is represented by a cross; irradiation without surgical treatment, by a dot. The degrees of regression of adenoids were as follows:

1. Large mass to clean nasopharynx.
2. Large mass to clean eustachian orifices.
3. Moderate mass to clean nasopharynx.
4. Moderate mass to clean eustachian orifices.
5. Small amount to clean nasopharynx.
6. Small amount to clean eustachian orifices.
7. Large mass to improvement.
8. Moderate mass to improvement.
9. Small amount to no change.
10. Moderate mass to no change.
11. Large mass to no change.
12. Unobserved treatments.

Five patients have received seven irradiations; 5 have received six and 21 patients five. (One of the latter still has unsatisfactory results.) The majority of patients, however, are considered well after three or four treatments, and a surprising number have noted definite improvement after a single treatment (chart 2).

Work being done at present indicates that radium applicator dosage described on page 476 may be considerably increased. It seems now that a dosage of "1 Gm. twenty-three seconds" is perfectly safe and that a dosage of "1 Gm. twenty-five seconds" is probably safe; it may be that eventually a dosage of "1 Gm. thirty seconds" may be used. All of the results described in this paper have been obtained with a dosage of "1 Gm. twenty seconds."

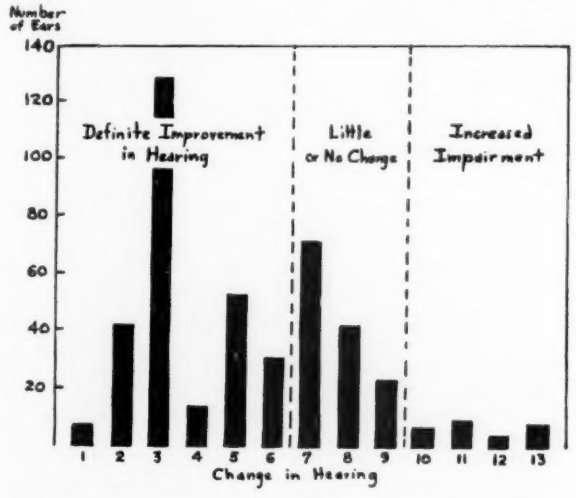

Chart 2.—Effect of treatment on hearing. The changes in hearing were as follows:

1. Severe impairment to normal hearing.
2. Moderate impairment to normal hearing.
3. Slight impairment to normal hearing.
4. Severe impairment to improvement.
5. Moderate impairment to improvement.
6. Slight impairment to improvement.
7. Slight impairment with little or no change.
8. Moderate impairment with little or no change.
9. Severe impairment with little or no change.
10. Slight impairment becoming worse.
11. Moderate impairment becoming worse.
12. Severe impairment becoming worse.
13. Totally deaf.

Adequate means of protection for the doctor administering radium therapy have not as yet been satisfactorily developed, but are being considered at the present time. It seems possible that when a doctor gives

radiation treatments frequently over a long period some damage may be done to his fingers or perhaps to his body unless additional precautions are developed in the future.

It would be a tragic mistake for any one to assume that irradiation in itself is the answer to any problem in otolaryngology. It cannot be overstressed that if any benefit is to be derived from radiation therapy, this therapy must be intelligently used in connection with those other forms of treatment which have proved to be of benefit in the past. The advantages of radiation lie in (1) the simplicity and the safety of its use and (2) the fact that lymphoid tissue which cannot be successfully attacked by surgical measures alone may be eliminated by irradiation.

The technical material in this article was provided by Dr. S. J. Crowe and Dr. Curtis Burnam.

6 East Eager Street.

DISTURBANCES OF THE ETHMOID BRANCHES OF THE OPHTHALMIC NERVE

With Description of a Syndrome Associated with Chronic Suppuration of the Olfactory Fissure

J. JEROME LITTELL, M.D.
INDIANAPOLIS

A SENSE of dissatisfaction as to the care of cephalic pain by giving attention to the sphenopalatine ganglion (Meckel's ganglion) and its connecting nerves led me to undertake studies of the ethmoid branches of the ophthalmic nerve. It is the purpose of this thesis then, first, to establish the importance of these nerves in rhinologic symptomatology and, second, to bring about recognition of mechanical defects of the olfactory fissure which produce chronic infection of this area, with pain stimuli being transmitted by these nerves. A symptom complex is produced which will be described as the syndrome of the olfactory fissure.

LITERATURE

It would seem that the study of irritations of the ethmoid nerve has not received the attention it deserves. If my search of the literature has been accurate, little emphasis has been placed on it, and that largely in the foreign literature.

In 1918 Sluder [1] emphasized the posterior innervation of the nose and the sinuses, with the sphenopalatine ganglion acting as a distributing center for pain stimuli. The vacuum frontal sinusitis which he described owed its presence to pathologic conditions in the middle meatus and infundibulum. Sensory innervation by the ethmoid nerves was not emphasized.

In 1925 Greene [2] described rhinologic headaches on the basis of pressure exerted by swollen tissues on sensory nerves or their termination. He mentioned one group of headaches "from anatomic variation

From the Department of Otorhinolaryngology, Indiana University School of Medicine.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society in June 1945.

1. Sluder, G.: Concerning Some Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1918.

2. Greene, D. C.: Rhinologic Aspect of Headaches and Cranial Neuralgias, Ann. Otol., Rhin. & Laryng. **34**:715-720 (Sept.) 1925.

in the nose with pressure contacts between septum and outer walls." He included them in Sluder's vacuum frontal sinus group.

In 1929 Dintenfass [3] stressed mechanical factors in the nose largely from septal deviations, with headaches produced by close approximation of the turbinates to the septum which may be increased by allergic swelling. He expressed the belief that the center of sensory distribution is Meckel's ganglion and failed to mention the branches of the nasociliary nerve.

In 1931 Charlin [4] presented 14 cases of what he described as the "syndrome of the nasociliary nerve," since known as Charlin's syndrome. The characteristics of the Charlin syndrome are:

1. One-sided acute rhinitis with excessive rhinorrhea.
2. Oculo-orbital neuralgia, usually extreme.
3. Lacrimation and acute inflammation of the external part of the eye, with possible keratitis and corneal ulceration.
4. Prompt relief and cure in a few days by simple cocainization of the anterior ethmoid region, which is angry and swollen.

This work is supported by numerous writers. Carlevaro [5] called attention to the particular interest which the syndrome has for oculists inasmuch as the symptoms are chiefly ocular, though it fails to respond to anything but intranasal care; he stated also that it is rare.

Limitations of space prevent a detailed discussion of the views of different writers. They are variously represented by Laskiewicz,[6] Lugli,[7] Chavira,[8] Majoros,[9] dos Reis,[10] Ayberk [11] and Della Vedova.[12]

Tremble,[13] in a good treatise on headache, mentioned "neuralgia" of the anterior ethmoid nerve in which relief of pain was secured by cocainization of the nerve at the point where it enters the nasal roof.

---

3. Dintenfass, H.: Headache from the Standpoint of the Rhinologist, Ann. Otol., Rhin. & Laryng. **38**:77-86, 1929.

4. Charlin, C.: Le syndrome du nerf nasale, Ann. d'ocul. **168**:86-102 (Feb.) 1931.

5. Carlevaro, G.: Sopra due casi di sindrome di Charlin, Ateneo parmense **11**:215-277 (July-Aug.) 1939.

6. Laskiewicz, A.: Sur les syndromes de Sluder et de Charlin, Rev. de laryng. **61**:117-129 (March) 1940.

7. Lugli, G.: Sindrome di Charlin, Riv. di clin. med. (supp. Margin. otolaryng.) **1**:89-96, 1940.

8. Chavira, R. A.: El síndrome del nervio nasal, Gac. méd. de México **72**:80-93 (Feb. 28) 1942.

9. Majoros, J.: Charlin Syndrome, Gyógyászat **80**:68 (Feb. 10) 1940.

10. dos Reis, G.: Un caso de síndrome de Charlin, Rev. brasil. de oto-rinolaring. **10**:709-715 (Nov.-Dec.) 1942.

11. Ayberk, N. F.: Burundan menseini alan bozi goz afetleri, Türk tib cem. mec. **5**:322-325, 1939.

12. Della Vedova, A.: Un caso dimonstrativo dei rapporti patologici nasooculari, Gazz. d. osp. **61**:446-449 (June 2) 1940.

In the literature of this country is an excellent review presented by McLaurin.[14] He stated that "nearly all headaches from intranasal pressure depend upon irritation of the sensory nerve endings in the membrane by some pressure and the effect that such irritation produces on the sphenopalatine ganglion or the anterior ethmoidal nerves."

Since the publications on Charlin's syndrome, Horton [15] has introduced a new entity, histaminic cephalgia. Its symptoms simulate in many respects those described by Charlin but differ in that they are paroxysmal, their distribution follows a vascular instead of a nerve course and they fail to respond to intranasal care (Horton [16]). It may be a part of the entity described by Fay [17] in 1932.

ANATOMIC CONSIDERATIONS

Limitations of time preclude a review of the anatomy of the ophthalmic nerve and its branches (fig. 1). One needs to recall that this nerve, through the branches shown in figures 1, 2 and 5, conveys sensory stimuli from the eyeball, the eyelids, the forehead, the root and a portion of the lateral surface of the nose, the ethmoid cells and, most important so far as this paper is concerned, the olfactory fissure. The close association of the nasal and the intraorbital fibers and their cells in the gasserian ganglion, the pons and the postcentral gyrus explains the similarity of symptoms from pathologic involvements of the two areas. It will be shown that pain in the eyeball can be and often is produced intranasally.

PATHOLOGIC OBSERVATIONS

The course of the anterior and posterior ethmoid nerves seems to invite inflammatory conditions of the nerve trunks or more particularly of their terminal branches (figs. 1 and 5). The nerves traverse bony canals where they are separated in a scant fashion from tissue which is capable of great inflammatory possibilities, acute as well as chronic. It seems reasonable that they might be subject to much more insult than the nerve tissue of the sphenopalatine ganglion, which is in much less delicate approximation to highly inflamed areas and certainly to

13. Tremble, G. E.: Headache of Nasal Origin, Canad. M. A. J. **50**:43-47 (Jan.) 1944.

14. McLaurin, J. G.: Headaches of Otorhinologic Origin and Certain Reflexes Caused by Fifth Nerve Irritation, Ann. Otol., Rhin. & Laryng. **50**:469-491 (June) 1941.

15. Horton, B. T.: Use of Histamine in Treatment of Specific Types of Headaches, J. A. M. A. **116**:377-383 (Feb. 1) 1941.

16. Horton, B. T.: Symposium: Head and Face Pain; Medicine, Tr. Am. Acad. Ophth. **49**:23-33 (Sept.-Oct.) 1944; personal communication to the author.

17. Fay, T.: Atypical Facial Neuralgia, a Syndrome of Vascular Pain, Ann. Otol., Rhin. & Laryng. **41**:1030-1062 (Dec.) 1932.

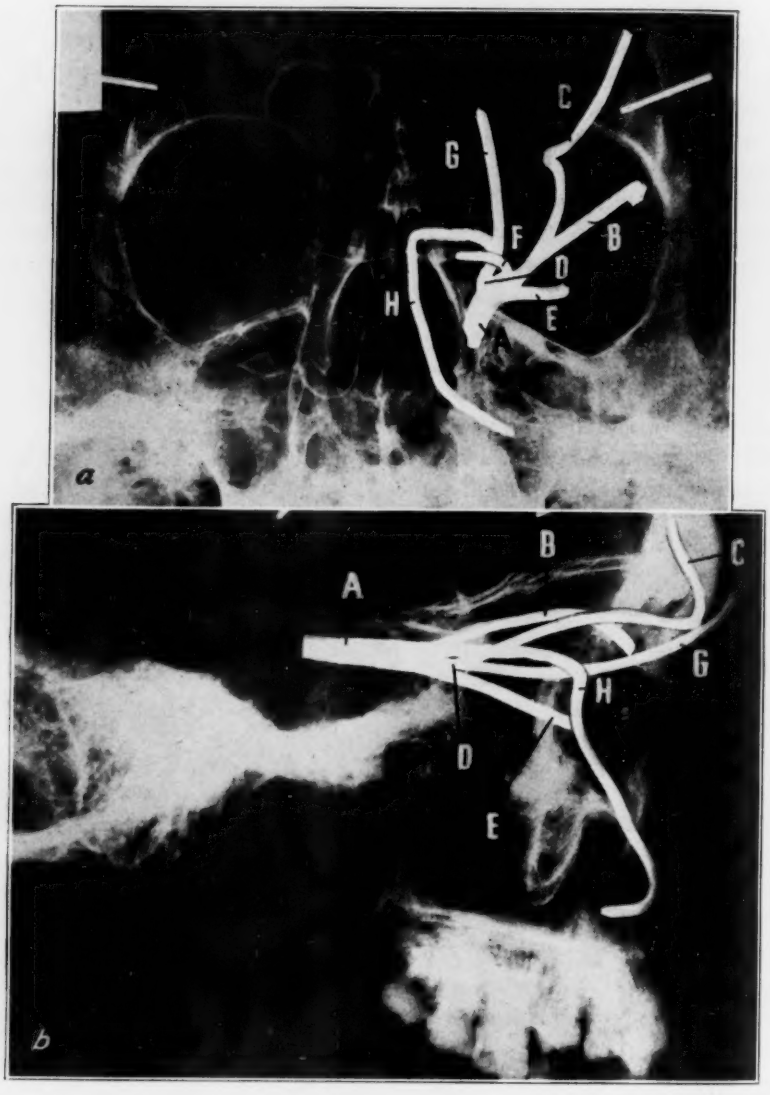

FIGURE 1, *a* and *b*

Distribution of the ophthalmic nerve. Different aspects (*a*, *b*, *c* and *d*) are shown on a dry skull. *A* indicates the ophthalmic nerve; *B*, the lacrimal division; *C*, the frontal division; *D*, the nasociliary division; *E*, ciliary nerves (usually three); *F*, the posterior ethmoid nerve; *G*, the infratrochlear nerve; *H*, the anterior ethmoid nerve, with termination as external nasal nerve.

FIGURE 1, *c* and *d*

areas which harbor infection because of mechanical blockades. The arrangement of these nerves (figs. 2 and 5) suggests to one that they hold dynamic sway over the narrow apex of the nose, the olfactory fissure and the ethmoid cells that have the burden of attempting to maintain normal function in this restricted area—an area which in my opinion determines the favorable or ill function of a substantial proportion of the problematic noses presented.

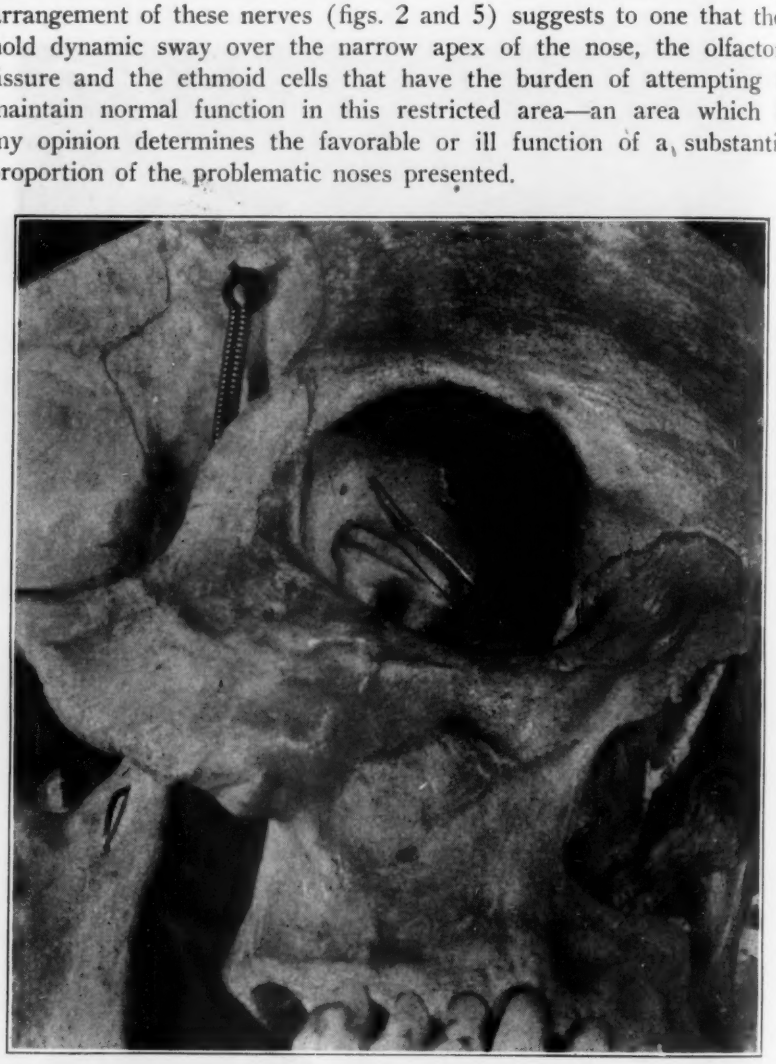

Fig. 2.—Medial aspect of the right orbit. The wires show an anomaly—three instead of two foramens at the line of articulation of the ethmoid and frontal bone. The mere location of these canals for passage of ethmoid nerves suggests the importance of these nerves in the symptom complexes referable to this area.

DEFECTIVE MECHANICS IN THE OLFACTORY FISSURE

As seen in figure 5*b*, the area between the septum and the crest of the upper overhang (Mosher [18]) of the middle turbinate body

18. Mosher, H. P.: A Speculum for Submucous Resection of the Septum, Laryngoscope **17**:88, 1907.

often presents little or no potential space. This crowding may be due to medial encroachment of the ethmoid cells, carrying the turbinal attachment with them, for they "have a tendency to grow steadily in all directions until deterred by compact bone" (Van Aylea [19]). It may be due to the tendency of intranasal tissue to occupy whatever space is available for growth (fig. 6). *It is on the narrow upper part of the nose that the premium on space casts the greatest pathologic burden.* Anterior to this tight area is almost invariably a more or less prominent tubercle of the septum, composed of the osseous portion of the anterior part of the perpendicular plate of the ethmoid bone and the cartilage of the septum (fig. *7a*). It is the thickest part of the normal septum (Mosher [18]) (fig. *7b*).

Whether excessive thickening of this structure is due to trauma and inflammation (Ingals [20]), rickets or faulty development (Freer [22]) is not clear. Its growth is unlimited by lateral pressure, as it stands

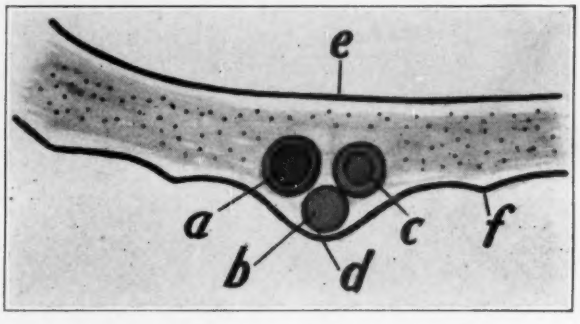

Fig. 3.—Representation of a sagittal section through the roof of the ethmoid cells after ethmoidectomy, showing the anterior ethmoid nerve, artery and vein enclosed in the roof of the intercellular septum in the fovea frontalis. It lies in the middle turbinate lamella. *a* indicates the vein; *b*, the nerve; *c*, the artery; *d*, the cut edge of the middle turbinate lamella; *f*, the cut edge of an intercellular septum.

almost 1 cm. from the nonerectile tissue on the frontal process of the maxilla, in the largest potential space in the nose, so far as my dissections show.

---

19. Van Alyea, O. E.: Ethmoid Labyrinth: Anatomic Study, with Consideration of Clinical Significance of Its Structural Characteristics, Arch. Otolaryng. **22**:881-902 (June) 1939.

20. Ingals, E. E.: Deflection of the Septum Narium, Arch. Laryng. **3**:291-299 (Oct.) 1882.

21. Footnote deleted by the author.

22. Freer, O. T.: The Anatomy of Deflections of the Nasal Septum, Tr. Am. Laryng. A. **34**:71-87, 1912.

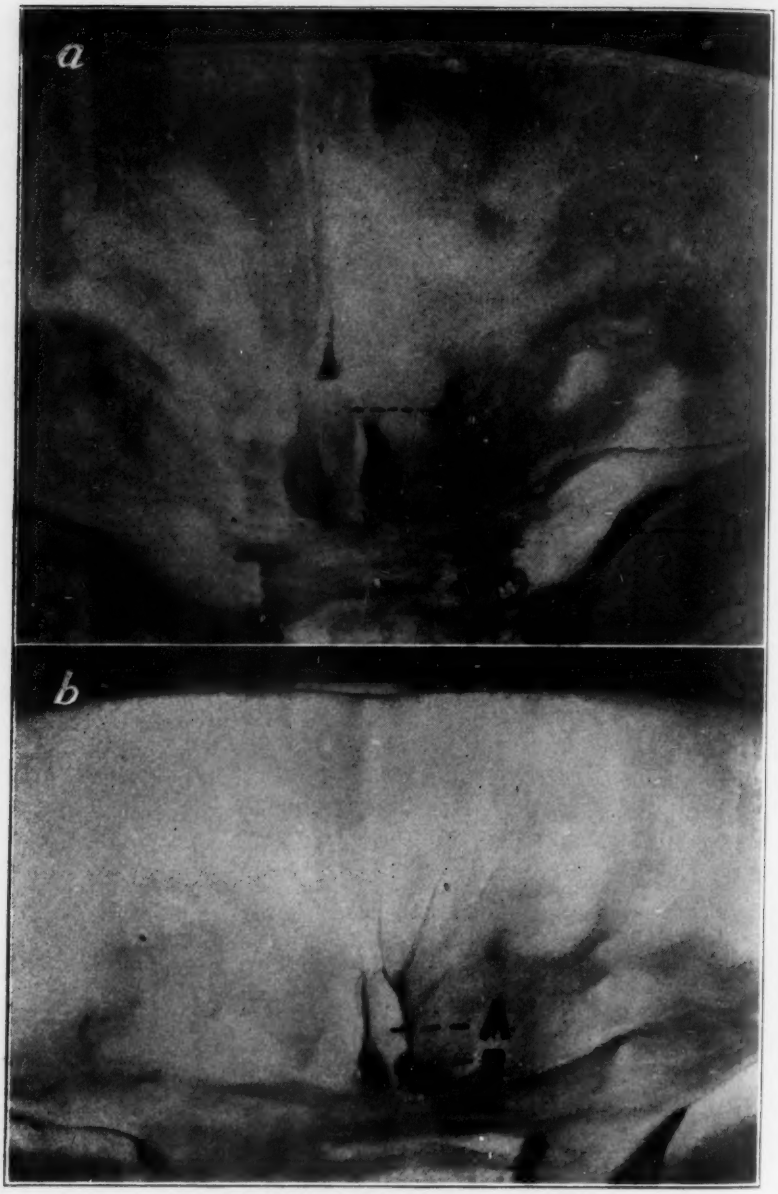

Fig. 4.—(*a*) Floor of the anterior cerebral fossa viewed from behind. *A* indicates crista galli; *B*, the slit through which the anterior ethmoid nerve and blood vessels pass into the olfactory fissure along with the olfactory nerves at *C*; *D*, the superior orbital fissure for passage of the ophthalmic nerve.

(*b*) Anomalous lateral development of the crista galli (*A*) making an anteroposteriorly directed canal for passage of the structures mentioned in (*a*).

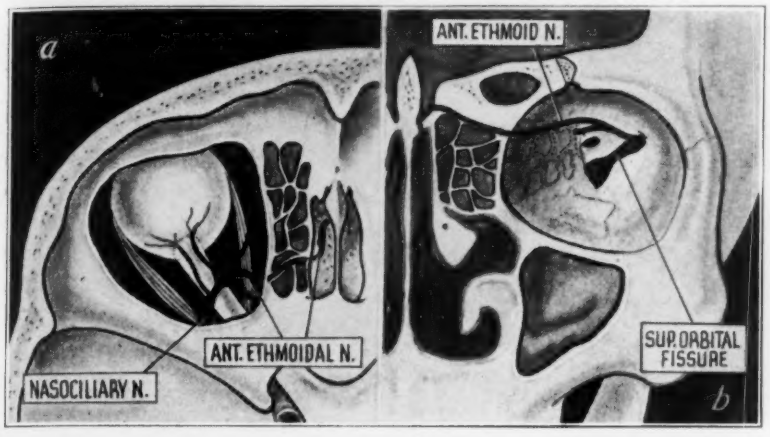

Fig. 5.—(*a*) Superior aspect of ethmoid nerves. The ciliary nerves to the eye and the posterior ethmoid nerves are not labeled. The ease with which ocular pain could be produced by irritation of the ethmoid nerve is suggested.

(*b*) Phantom and cut section through the left orbit showing the intranasal portion of the anterior ethmoid nerve intimately connected with the anterior portion of the olfactory fissure. The distribution of the posterior ethmoid nerve is similar but more posterior (according to O. V. Batson in a personal communication to the author). Note the anaerobic pocket formed in the left retrotubercular area.

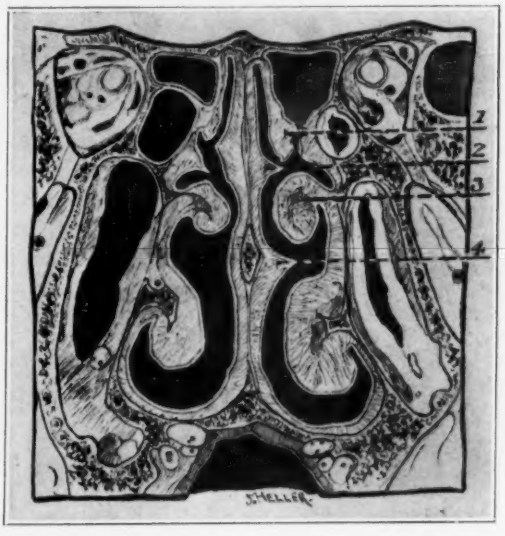

Fig. 6.—An illustration from Sluder's [1] book showing the tendency of intranasal tissue to occupy available space. Note the spurs (*2* and *4*) above and below the middle turbinate (*3*). *1* indicates the upper turbinate.

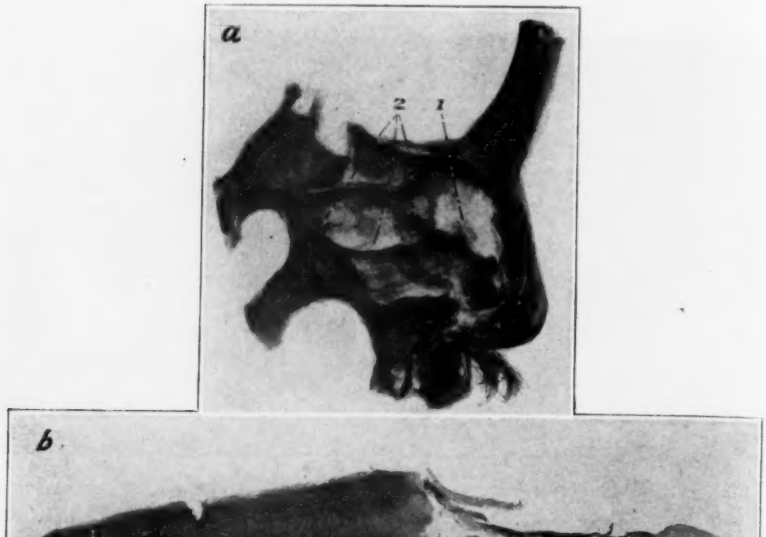

Fig. 7.—(*a*) An illustration from Sluder's [1] book. *1* indicates the well marked tubercle; *2*, the deeply marked imprint of the entire middle turbinate. The septum was not deflected to either side. Retrotubercular pocket shown.

(*b*) Section through a septal tubercle removed from a patient showing chronic suppuration of the olfactory fissure following blockade. *1*, cartilaginous portion; *2*, decalcified perpendicular plate of the ethmoid bone.

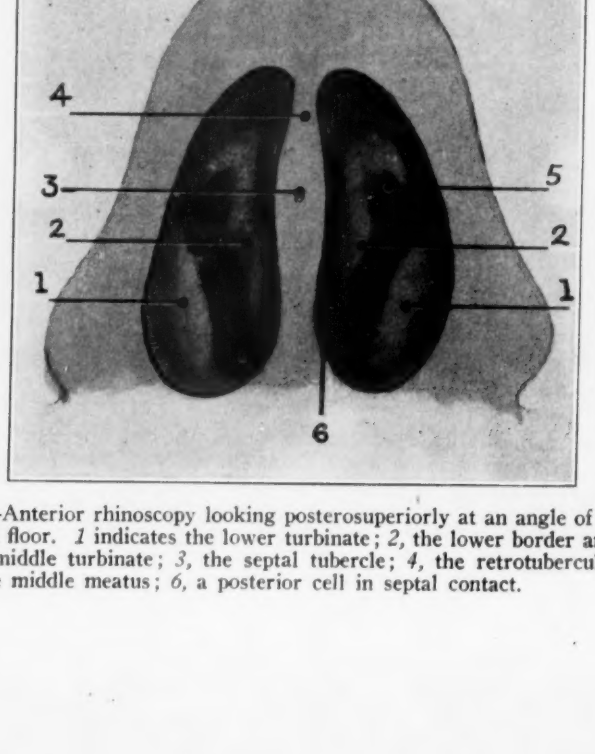

Fig. 8.—Anterior rhinoscopy looking posterosuperiorly at an angle of 45 degrees to the nasal floor. *1* indicates the lower turbinate; *2*, the lower border and anterior tip of the middle turbinate; *3*, the septal tubercle; *4*, the retrotubercular contact area; *5*, the middle meatus; *6*, a posterior cell in septal contact.

PATHOLOGIC ALTERATION IS NOT REVEALED BY ANTERIOR RHINOSCOPY

The area above and behind this thickened portion of the septum (could one coin the word retrotubercular space?) is often invisible to anterior rhinoscopy (fig. 8) and cannot be visualized until after submucous operation has allowed a view of it. *Its pathologic states are not apparent, then, unless they are strongly suspected of being present when certain symptom complexes present themselves and the causes are hunted down.* In cases in which adequate study has justified surgical approach to this area one is rewarded by the observation of a tight anaerobic area between the anterior third of the middle turbinate body, the area immediately above it and the septum. This area is angry, will not blanch out with the epinephrine which has been used to prepare it for surgical treatment and contains a gross amount of mucopurulent or almost pure purulent discharge (fig. 9*a*). I feel that this area when walled off in this closed manner is capable of producing as many symptoms as a blocked sinus, for after all it is an enclosed area lined with the same mucous membrane (fig. 9*b*). It is easily possible that it might produce symptoms of an exaggerated variety for the following reasons:

1. This area is extremely vascular, serving as it does to give passage to the diverging branches of the anterior ethmoid artery and vein. It contains more cavernous tissue than any other part of the middle turbinate (Sluder [1]).
2. The terminal intranasal branches of the anterior ethmoid nerve diverge here from the foramen in the cribriform plate.
3. The tissue in this area is five times as sensitive as that within the sinuses themselves and is comparable in sensitiveness only to the tissue around sinus ostiums.[23]

The air currents which, as Proetz [24] has so well shown, should pass in an upward direction over the area of the cribriform plate have been prevented from doing so by this blockade. This closed anaerobic area becomes more or less a cesspool for micro-organisms and is then quite adequate to produce many symptoms. These organisms are largely pyogenic, although their leukocytic product is not seen within the nose, being recovered only in the pharynx, where its presence accounts for much otherwise unexplained disease. I feel that this condition is most common.

AIR CURRENT DEMONSTRATION

In order to demonstrate whether inspired air currents reach this constricted area or are deflected laterally by the septal tubercle in cases

23. Nash, S.: Functional Diseases of the Nose, Course at Academy of Ophthalmology and Otolaryngology, October 1944.

24. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941, p. 114.

Fig. 9.—(*a*) Medial surface of the middle turbinate body in the retrotubercular area, showing chronic infection identical in character to that shown in (*b*). (*b*) Ethmoid cell lining, removed in toto, showing contained cellular elements.

in which pathologic conditions are present, powder was introduced into defective noses. Sulfadiazine powder was insufflated into the nose by a method previously described by me [25] and directed upward and backward in the direction described by Proetz as that taken by air currents. Patients were also asked to breathe in finely divided powder from a bag. In each case, after the septum was straightened, the distribution of the powder was noted. No powder had reached the aforementioned comparatively anaerobic retrotubercular area.

DIAGNOSIS

The same mechanics by which air currents (which tend to travel a straight course unless impeded) are prevented from properly reaching

Fig. 10.—(*a*) Mrs. D. four days after section of the right anterior ethmoid nerve and vessels. The wound is invisible now. Below the incision there is excoriation from adhesive tape.

(*b*) Mrs. S. before a similar operation for relief of the syndrome of the olfactory fissure. The pain in the left eye was so severe as not to be relieved by a preoperative hypodermic injection of H. M. C. # 1 (a mixture stated to contain hyoscine, morphine and cactine). It ceased during operation on intraorbital cocainization of the ethmoid nerve and except for slight postoperative soreness has not returned.

the roof of the nose at this point also present visualization of this point by anterior rhinoscopy (fig. 8). The septal tubercle projects itself obstructively in front of the middle turbinate for 8 to 12 mm. (Mosher [18]). Inspection of this nasal cavity, concerning the faulty

25. Littell, J. J.: Undiluted Sulfonamide Drugs in Sinus Surgery, J. Indiana M. A. **36**:250-252 (May) 1943.

function of which the patient complains, may reveal no evident pathologic change. The part of the middle turbinate which is visible may not be congested and may shrink down readily to an almost normal pink, and no pus is seen. There is no blockade or congestion in the middle meatus, which is well ventilated by the air currents thrown its way by the turbercle. Disease in the larger sinuses has been eliminated and the posterior ethmoid area is seen to be free of abnormality. Unless the physician is aware of the possibility of pathologic developments in the unseen anterior portion of the olfactory fissure, he will dismiss the case as one without foundation in organic disease. It is possible for a posterior cell area impinging against the septum in the "struggle of the ethmoids" (Fenton?) to produce difficulties of a similar nature, with the symptoms being transmitted by way of the smaller posterior ethmoid branch. The latter type of blockade is important and frequent but is less often overlooked than that of the retrotubercular area, because it is often associated with definite septal deviation. Furthermore, this posterior high blockade may produce recognizable suppurative posterior ethmoiditis, as the cell ostiums are located there. It accounts, I believe, for the ill function which sometimes exists on the side of the nose opposite to a septal deviation. The contact area is invisible, behind a hypertrophied middle turbinate.

CLINICAL PICTURE

I agree with other observers that the typical dramatic picture of Charlin's syndrome is rare. I can recall 3 cases of this type in my practice. There have been many more of a milder type, without the extreme rhinorrhea and lacrimation and with simpler forms of congestive phenomena in the eye as well as in the nose. These have cleared up within a short time following mild shrinking and cocainization of the anterior ethmoid region. I have shared the astonishment of all previous observers that the relief of the ocular symptoms in these cases has been so dramatic.

THE SYNDROME OF THE OLFACTORY FISSURE

There are other cases, however, which through sacrifice of the aforementioned dramatic quality lose nothing in importance, because of their great frequency. They exhibit a symptom complex which I think of as the syndrome of the olfactory fissure. More uncommonly, if the process is acute and active, the pain is more widespread, and the patient may be unable to localize it. The characteristic symptoms, however, are unilateral or bilateral pain of a dull and continuous character throughout the entire sensory distribution of the ophthalmic nerve. The aching is deep in or behind the eyeball, in the frontal region and sometimes at the root of the nose. Occasionally, pain is referred to an area

centering 7 cm. posterior and 7 cm. inferior to the external auditory meatus or to the region of the ear. These areas are not particularly tender to pressure, nor are they trigger zones such as one finds in the neuralgic type. The localization is supported to a large degree by the experiments of McAuliffe, Goodell and Wolff [26] in which pain was produced by faradic stimulation. Symptoms of irritation of the sympathetic elements are absent (e. g., lacrimation and rhinorrhea). The nose is usually stuffy on the affected side, although the patient may be unaware of it. The symptoms may alternate from side to side, as the nasal passages become alternately stenosed from engorgement of tissues.

This syndrome is characteristically present over long periods—perhaps most of the patient's life—and he has often found it necessary to live over and above it, accepting it as his lot. It is activated by any condition which predisposes to engorgement of intranasal tissues. The order of frequency from my observation is as follows: (1) damp or changeable weather (a frequent occurrence in this latitude), (2) exposure and chilling of the body surface, (3) allergies, (4) fatigue, (5) emotional disturbances, (6) glandular dyscrasias, (7) gastrointestinal disorders and faulty dietary habits, (8) alcohol and (9) chronic infection of any sinus on the affected side.

Associated with these periods of activity one almost invariably finds fatigue and often generalized aching; the latter may be vague or localized in the form of periarticular fibrositis.

The syndrome of the olfactory fissure, then, includes:

1. Pain or aching of a nonparoxysmal nature in or about the eye, relieved as outlined in the following section.
2. Nasal stuffiness, worse on the affected side.
3. Postnasal discharge, perhaps evidenced only by an inflamed lateral pharyngeal band on the affected side, more evident on gagging.
4. Systemic symptoms of absorption of a toxin.

EFFECT OF DIVISION OF THE ANTERIOR ETHMOID NERVE

It seemed desirable to prove whether or not the pain in a typical case of the syndrome of the olfactory fissure was conveyed by the ethmoid nerves or by the sphenopalatine route.

Mrs. O (fig. 10a), aged 54, a phlegmatic country woman who was an outpatient at Indiana University Medical Center, gave a history of pains behind and in the right eye and the right supraorbital region with occasional reference to the right ear. These had been more or less continuous all her life but in the last few years had become progressively worse to the point that she was often unable to work. They were characteristically worse in damp weather and

26. McAuliffe, G. W.; Goodell, H., and Wolff, H. G.: Experimental Studies on Headache. A. Research Nerv. & Ment. Dis., Proc. (1942) **23**:185-208, 1943.

when more active were accompanied by fatigue and generalized aching. All studies gave negative results (including that of a nasal smear for eosinophils) except that there was generalized haziness of the ethmoid and maxillary sinuses and intranasal defects. Here there was a moderate high anterior septal deviation, made more prominent by the tubercle, but no pus or definite inflammation was seen. The middle meatus was not abnormal. Behind the tubercle an area of septal contact was felt. This was cocainized in the manner described in the following section, and the pain was substantially relieved in fifteen minutes. Repetition produced similar results. General measures had been adequately tried without benefit. On Aug. 27, 1944, through an external incision similar to that used in the external frontoethmoid operation, but without disturbing the trochlea, the anterior ethmoid nerve, artery and vein were divided by electrocoagulation as they entered the anterior ethmoid foramen. Instrumental pressure on the posterior ethmoid nerve produced retrobulbar pain similar to that of which the patient complained. As no posterior intranasal pressure existed, this nerve was not divided.

After a few days the patient's pain was gone. She expressed herself as being especially grateful for the relief of the boring pain in and behind the eye. The tactile sense of the anterior two thirds of the middle turbinate and the septum and that of the olfactory fissure were much reduced. On October 25 she described her health as excellent, with return of her old energy and absence of pain.

I am aware of the fact that her relief might be explained partly on a psychic basis, although I think she is not the type. Also the diminution of the blood supply to the part might relieve congestion in that area, with improvement in symptoms, contrary to the view expressed by Proetz [27] that ischemia may be productive of pain. It seems logical, however, to suppose that the pain stimuli from the tight infected area which were conveyed over this nerve were interrupted by its division.

A second patient, Mrs. A. S., in the same clinic (fig. 10*b*), with an almost identical picture except for a shorter duration of symptoms (seven years) and radiologically normal ethmoid sinuses was similarly relieved. For six months she had had a numb feeling down the homolateral arm and fingers on the ulnar side. This disappeared four days after operation.

The future course of the complaints of these patients will be watched with interest, as there is no assurance that their relief will be permanent. It is possible that this comparatively minor procedure may have value in intractable cases of this kind in which there is no response to intranasal measures.

DIAGNOSIS

The simple criterion of this symptom complex is the relief of symptoms on shrinking with a mild anesthetic agent (i. e., a 4 per cent cocaine salt preferably in naphthylmethylimidazoline hydrochloride [privine], neo-

27. Proetz, A. W.: Symposium: Head and Face Pain; Otolaryngology, Tr. Am. Acad. Ophth. **49**:18-22 (Sept.-Oct.) 1944.

synephrine or a 1 per cent solution of an ephedrine salt). A fine applicator, the last half inch angulated, delicately cotton tipped and the cotton impregnated with the shrinking agent, is introduced into this tight area repeatedly. The Proetz suction displacement may be useful if obscure ethmoiditis is present (Shambaugh [28]). If some measure of relief—it may even be complete—ensues within fifteen minutes to several hours, and this process can be repeated on numerous occasions to avoid coincidence, one may feel that the suspected area is productive of headache. If one suspects psychic relief, a placebo may be tried; but the careful rhinologist is hard to deceive.

One is aware that ethmoiditis may produce similar symptoms, as this also involves irritation of the ethmoid nerves. It may as a matter of fact, but need not, accompany chronic suppuration in the olfactory fissure. Diffuse ethmoiditis might cause stimulation of the sphenopalatine branches also with some additions to the syndrome described. Our diagnostic problem might in that case receive roentgenologic support. But I have learned not to base a diagnosis on roentgenologic findings, as they do not reveal this pathologic condition of the olfactory fissure. The patient may demonstrate the described symptom complex and be entirely relieved of it by suitable measures although the ethmoid sinuses are clinically and roentgenographically normal.

PERMANENT RELIEF MEASURES

There are cases in which attention to and elimination of the predisposing factors mentioned, together with occasional use of intranasal drops or resort to intranasal treatment, may keep the patient comfortable. Attention to hyperfunction of the nose with the attendant causes (Nash [23]) may serve to relieve a certain number of patients. Or careful history taking and guidance as outlined by Roberts [29] may be helpful (in many of the cases in his series this pathologic condition of the olfactory fissure might be reasonably shown with nasociliary pain in some and in some without it). There is a definite group, however, in which surgical correction of bad mechanics is necessary, and if properly conceived and executed it will rarely be disappointing.

An adequate removal of all possibility of inflammatory blockade must be contemplated. The first important step is a meticulously performed high and anterior submucous resection. The Cullom [30] forceps is invaluable here. If there is some deviation in addition to the projecting tubercle so that the correction produces at least 3 mm. of free space

28. Shambaugh, G. E., Jr.: Obscure Low Grade Chronic Infections of Ethmoid Sinuses, J. A. M. A. **112**:1126-1129 (April 1) 1939.

29. Roberts, S. E.: A New Sinus Syndrome, Tr. Am. Acad. Ophth. **49**:177-193 (Jan.-Feb.) 1945.

30. Cullom, M. M.: Personal communication to the author.

in the inflamed area, and if the tissue of that area is not too hyperplastic from long severe disease, this may be sufficient. If, however, these conditions cannot be met, or if there is a substantial amount of chronic ethmoiditis present, I have found that disappointments are anticipated and prevented by doing ethmoidectomy at the same time with or without removal of the anterior half of the middle turbinate body. The greater the pathologic alteration the more complete the operation should be. I believe that half-hearted ethmoidectomy is as productive of later trouble as partial tonsillectomy. The roof of the nose must be free, with no suggestion of contact of tissues. Air *must* reach the olfactory fissure. Fomon's [31] insistence on this being achieved through rhinoplastic procedures is a contribution.

The external operation seems rarely necessary for the correction of "sinus headache," as the pain can be stopped almost invariably by adequate, properly conceived and executed intranasal measures. Failure to be critical of the area along the nasal roof may account for some of the disappointments in this work, such as that reported by Faulkner,[32] although, after not a little experience in this field, I feel in hearty accord with him when he says "there is not a more spectacular operation in the field of surgery than intranasal ethmoidectomy."

CONCLUSIONS

1. The ethmoid branches of the nasociliary nerve are important in rhinologic symptom complexes. Mechanical defects of the olfactory fissure produce chronic infection of this area and a symptom complex which might be termed the syndrome of the olfactory fissure.

2. In the literature emphasis has been largely on the sphenopalatine ganglion and its connections as the pathway for stimuli in most of the pain of rhinologic origin. The studies reported here suggest that in many cases such pain may be explained instead on the basis of irritation of the ethmoid nerves.

3. Charlin's syndrome is an explosive manifestation of all the elements of the nasociliary nerve.

4. The anatomic distribution of the intranasal branches of the nasociliary nerve, as well as the surgical experiments with their division described in this paper, suggests that they deserve prominent mention in the field of cephalic pain of intranasal origin.

5. Approximation of the tissues of the olfactory fissure produces the familiar chronic inflammatory changes which one sees within the

31. Fomon, S., in postgraduate instruction at Manhattan General Hospital, New York.

32. Faulkner, E. R.: End Results of Intranasal Operations on the Ethmoid, Frontal, and Sphenoid Sinuses, Arch. Otolaryng. **30**:716-720 (Nov.) 1939

sinuses themselves and for the same reason. The membrane of this area, however, is five times as sensitive as far as production of pain goes, as that within the sinuses, and therefore disease here and the correction of it is most important.

6. The syndrome of the olfactory fissure may be just as incapacitating as that of chronic ethmoiditis, which it resembles. The condition which produces it often escapes detection, however, as the area of the olfactory fissure is not visible because of septal projections, and the sinuses themselves may appear normal.

7. The diagnosis is based on careful clinical observation and therapeutic tests.

8. The results of proper treatment are eminently gratifying.

NOTE.—As of April 18, 1946, the relief exhibited by the 2 patients whose symptoms were recited in the foregoing pages has been maintained. Two other patients have been treated with similar results.

AERO-OTITIS MEDIA IN PRESSURE CHAMBER "FLIGHTS"

CAPTAIN ALFRED T. LIEBERMAN
MEDICAL CORPS, ARMY OF THE UNITED STATES

SINCE Armstrong and Heim [1] published the original description of aero-otitis media in 1937, much has been written about this relatively new morbid process. Under the impetus of World War II and the vast increase in flying which has accompanied it, the problem of aero-otitis media has come to assume much more importance in aviation medicine than ever before. At the present moment its chief importance lies with the military in that all Army and Navy flying personnel are subject to its disabling consequences; however, the scope of the problem will widen with the advent of peace and the dawning of a new and greater era of commercial aviation.

The present article is a report of observations on aero-otitis media that occurred as a result of high altitude "flights" in pressure chambers. "High altitude flights" in pressure chambers do not exactly duplicate actual flying; nevertheless the simulation of the physical conditions is such that chamber "flights" are used for testing and screening flying personnel and matériel throughout the Army Air Forces.

Certain observations regarding the pathogenesis of aero-otitis media seem to be well established. Among these are:

1. A change in barometric pressure in the middle ear is the "sine qua non" of the process.
2. Inability to ventilate the middle ear either voluntarily or involuntarily permits the traumatizing effect of the changing barometric pressure to be exerted to the fullest extent.

There remain, however, other factors whose relationship to aero-otitis media are poorly or incompletely understood. These include: (1) the effect of various "altitudes" from which "descents" are made in the chamber and the rate of "descent," (2) the status of the nasopharynx and the pharyngeal orifice of the eustachian tube, (3) the effect of a subsiding or a subclinical infection of the upper respiratory tract, (4) the effect of low temperature and (5) the effect of previous flying experience. Observations on the foregoing points in their relationship to the occurrence of aero-otitis media form the basis of this report.

1. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. **109**:417-421 (Aug. 7) 1937.

All observations were made on subjects undergoing "high altitude flights" in low pressure chambers at the Aero-Medical Laboratory, Wright Field, Dayton, Ohio, early in 1943. Studies were carried out on 167 subjects participating in a total of 330 chamber "flights." The "flights" were conducted in two types of chambers: one in which the temperature was maintained at —40 F. and a second chamber in which "flights" were conducted at room temperature. The "flights" were of three different types: (1) a three hour run in which subjects were "flown" to a simulated altitude of 35,000 feet (10,600 meters) and remained there for three hours; (2) a cold run, i. e., a one hour run at 30,000 feet (9,000 meters) at —40 F.; (3) a simulated free fall in which the subject was "dropped" from 30,000 to 18,000 feet (5,500 meters) without oxygen in a period of about fifty seconds.

DIAGNOSTIC CRITERIA FOR AERO-OTITIS MEDIA

Although the syndrome of aero-otitis media is commonly encountered by workers associated with flying personnel, with men taking altitude chamber tests and with deep sea divers, a certain amount of confusion has crept into the consideration of the problem because of the failure to state definitely the criteria on which the diagnosis is made. For the purposes of this report the term "aero-otitis media" will be used throughout and is based on certain rigid criteria. The criteria used are the presence of subjective symptoms plus pathologic changes in the tympanic membrane or middle ear as revealed by otoscopic examination. Symptoms were variable and ranged from a "clogging" or "full" sensation in the ear to acute, severe pain which required "leveling off" in the "descent" or "reascending" to clear the ears. Pathologic changes varied from simple injection along the malleus to diffuse hyperemia and presence of fluid or blood in the middle ear cavity. In 1 case of the series a ruptured tympanic membrane was encountered.

INCIDENCE OF AERO-OTITIS MEDIA

In this study 167 subjects participated in a total of 330 "flights," an average of 1.98 "flights" per subject. Some subjects experienced aero-otitis media on more than one "flight"; 43 subjects had 55 episodes of aero-otitis media. It will be seen in chart 1 that, based on the previously described criteria for aero-otitis media, there were 55 cases of aero-otitis media in 330 "flights," a percentage of 16.6. In the same number of "flights," however, routine otoscopic examination of each subject immediately after a "flight" showed that in addition to the 55 cases in which symptoms plus objective findings of aero-otitis media were recorded there were 37 cases in which pathologic changes were observed in the tympanic membrane and middle ear though the patient had no symptoms. The total number of cases, therefore, in which

abnormal changes were observed in the tympanic membrane and the middle ear was 92 in 330 "flights," a percentage of 27.9.

It is interesting to note that in the entire series of 330 "flights" only 3 subjects had symptoms with no pathologic alterations of the tympanic membrane or the middle ear. A possible explanation is that these subjects had either a low threshold of pain or a high degree of suggestibility. It is my firm conviction that difficulty in ventilating the middle ear sufficient to cause symptoms will always produce objective changes in the tympanic membrane and the middle ear.

EFFECTS OF ALTITUDE, RATES OF DESCENT AND LOW TEMPERATURE ON INCIDENCE OF AERO-OTITIS MEDIA

Because of the varied types of chamber "flights" conducted at the laboratory, an opportunity was afforded to observe the influence of varying conditions on the incidence of aero-otitis media.

The incidence of symptoms and objective findings in each of the three different types of chamber "flights" is shown in chart 2. In the "three hour run," symptoms plus pathologic findings of aero-otitis media occurred in 15.8 per cent of the "flights"; findings, in 29.5 per cent. In the "cold run," symptoms plus pathologic findings of aero-otitis media occurred in 12.5 per cent; findings, in 21.6 per cent. In the "free fall," symptoms plus pathologic findings of aero-otitis media occurred in 19.7 per cent; findings, in 30.6 per cent.

On analyzing these data, several factors seem worthy of comment:

1. A higher incidence of aero-otitis media (symptoms and findings) and of pathologic findings occurred in the "free fall" as compared with the "cold run" and the "three hour run." This would be expected since a change in altitude from 30,000 to 18,000 feet is brought about in approximately fifty seconds. This entails undergoing a pressure differential from 225.6 mm. to 379.6 mm., or 154 mm., of mercury in a very short period. In every person, therefore, who has difficulty in ventilating his middle ear either voluntarily or involuntarily, an ear block of varying degree is apt to develop.

2. Apparently low temperature in itself has no appreciable effect on the production of aero-otitis media. A lower incidence of aero-otitis media and of pathologic findings was observed in subjects undergoing "runs" in the cold chamber than in either of the other two types of "flights." This lower incidence may be due in part to the fact that the altitude from which the "cold run" was conducted was 30,000 feet, whereas the "three hour run" was conducted at 35,000 feet. This means that persons "descending" from a "cold run" undergo a pressure differential somewhat less than that in "descent" from a "three hour run."

3. All three types of runs produced pathologic changes in the tympanic membrane and the middle ear in a high percentage of subjects, although not always with associated symptoms. It seems reasonable to infer, therefore, that slight additional barometric trauma would have caused pain in many of the subjects who were asymptomatic but had pathologic changes. The relatively high proportion of 27.9 per cent with pathologic findings seen in chart 1 must be considered as the potential aero-otitis media group, and it is to this group that all methods of minimizing the deleterious effects of barotrauma should be directed.

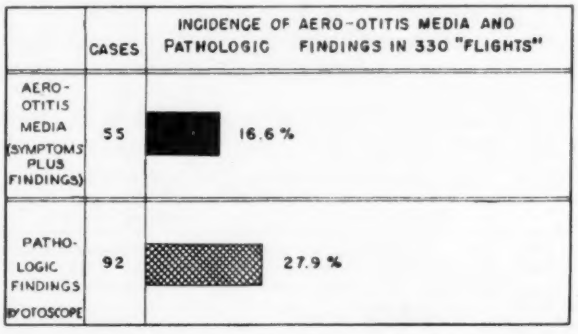

Chart 1.—Over-all incidence of aero-otitis media (symptoms plus otoscopic pathologic findings) and total incidence of otoscopic pathologic findings in 330 chamber "flights."

THE STATUS OF THE NASOPHARYNX AND OF THE ORIFICES OF THE EUSTACHIAN TUBES IN RELATION TO THE INCIDENCE OF AERO-OTITIS MEDIA

In an effort to understand better the role of the nasopharynx and the eustachian tubes in relation to aero-otitis media, the nasopharynx and the ostiums of the eustachian tubes were examined with a nasopharyngoscope in 129 men. In 12, or 9.3 per cent, of these the ostiums of the eustachian tubes were found completely or nearly occluded with lymphoid tissue. In those in whom they could be recognized at all the ostiums appeared as mere slits, while in others they were completely hidden from view by hypertrophied lymphoid tissue arising from a main central mass in the nasopharynx and from the fossae of Rosenmüller. Although the remaining 117 subjects examined with the nasopharyngoscope were classified as having unobstructed ostiums, a large percentage had masses of lymphoid tissue filling the fossae of Rosenmüller and compressing the ostiums of the eustachian tubes but not obscuring them.

A comparison of the group with obstructed and the group with nonobstructed ostiums in regard to incidence of aero-otitis media shows that in 20 "flights" participated in by the 12 subjects with obvious

obstruction of the eustachian tubes aero-otitis media occurred eight times, an incidence of 40 per cent. In 218 "flights" participated in by the 117 subjects without obvious obstruction of the eustachian tubes, aero-otitis media occurred thirty-three times, an incidence of 15.1 per cent. In terms of the individual susceptibility to aero-otitis media with obstructed and with nonobstructed ostiums, 6, or 50 per cent, of the 12 subjects with obstruction had aero-otitis media, whereas 16, or 13.7 per cent of the 117 subjects without obvious obstruction had aero-otitis media.

THE EFFECT OF SUBSIDING OR SUBCLINICAL INFECTIONS OF THE UPPER RESPIRATORY TRACT ON THE INCIDENCE OF AERO-OTITIS MEDIA

It is generally accepted that the presence of an acute infection of the upper respiratory tract predisposes flying personnel to aero-otitis media and aerosinusitis. For this reasons, no subject who volunteered a history

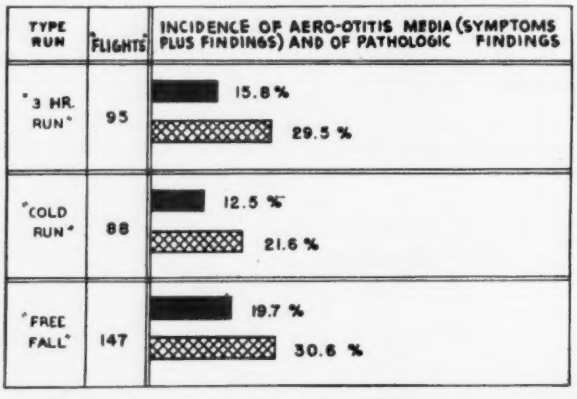

Chart 2.—Incidence of aero-otitis media (symptoms plus pathologic findings) and total incidence of pathologic findings noted with different types of chamber "flights." The solid black block represents aero-otitis media (symptoms plus findings); the cross-hatched block, otoscopic pathologic findings.

of an acute infection of the upper respiratory tract was permitted to go into the chamber. Similarly, no subject with a history of subsiding infection of the upper respiratory tract in whom clinical evidence of acute infection could be seen was permitted to participate in the chamber runs.

Among 165 of the subjects questioned, 60 gave a history of "just getting over a cold," "a nose stopped up," "a slight cold," etc., but no objective evidence of acute infection could be seen on examination. The remaining 105 subjects gave an unequivocal answer of "no" in response to questioning regarding a recent cold. It is of interest to note the percentage of subjects with aero-otitis media in the two groups

(chart 4). Of those with a history of a recent cold, 23.3 per cent had aero-otitis media, whereas 26.6 per cent of the subjects with no history of a cold had aero-otitis media. From such observations it seems justifiable to permit a subject with a history of recent infection of the upper respiratory tract to participate in chamber runs if no objective evidence of acute infection is present.

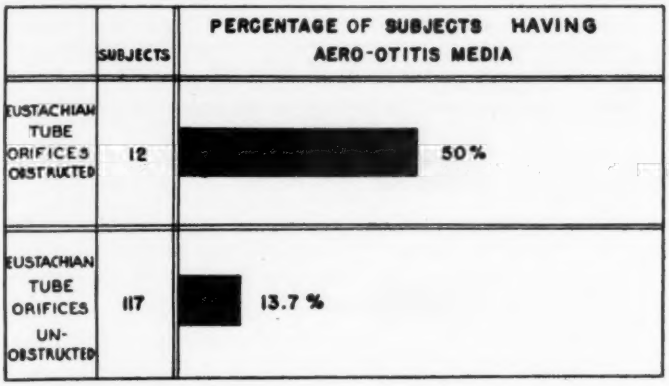

Chart 3.—Incidence of aero-otitis media in subjects with orifices of the eustachian tubes obstructed and subjects with these orifices unobstructed.

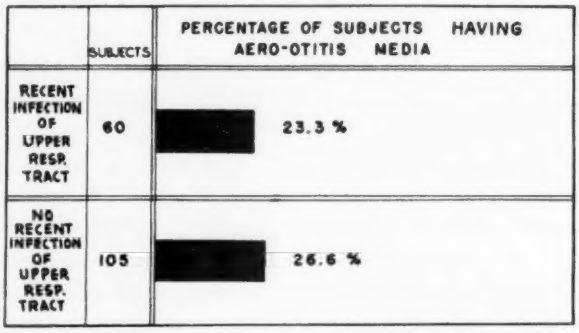

Chart 4.—Incidence of aero-otitis media in subjects who stated that they had had a recent infection of the upper respiratory tract and in subjects who definitely denied having had such infection.

THE EFFECT OF FLYING EXPERIENCE ON THE INCIDENCE OF AERO-OTITIS MEDIA

The role that experience and practice in clearing one's ears plays in the prevention of aero-otitis media is imperfectly understood. It has been observed frequently that aero-otitis develops in many subjects because of lack of instruction as to how to clear their ears properly. Nevertheless, aero-otitis media occurs even in the most experienced of flying personnel when undergoing "high altitude flights" in the pressure chamber.

In the group of subjects with whom the present report is concerned, there were 135 men who were rated pilots, men whose experience included test piloting, pursuit, dive bombing and heavy bombardment flying. The remaining 32 men had had no flying or pressure chamber experience. Aero-otitis media developed in 35 (25.9 per cent) of the 135 men in the group with flying experience and in 8 (25 per cent) of the 32 men in the group with no flying experience.

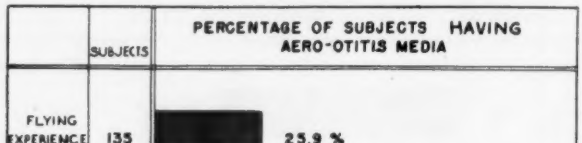

Chart 5.—Incidence of aero-otitis media in subjects with flying experience and subjects with no flying experience.

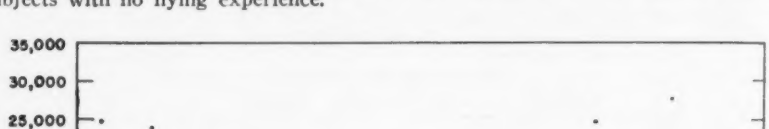

Chart 6.—Scattergraph showing altitude at which onset of symptoms occurred in 46 men with aero-otitis media.

The absence of any significant difference in the incidence of aero-otitis media in the two groups emphasizes the complexity of the various factors involved in the production of aero-otitis media.

OBSERVATIONS REGARDING ALTITUDES AT WHICH ONSET OF SYMPTOMS OCCURRED

Since change in barometric pressure is generally conceded to be the one common factor in the cases of aero-otitis media, it is important to learn as much as possible about the relationship of barometric pressure to aural symptoms. Armstrong [1] stated: "At about 60 mm. of mercury negative pressure the pain in the ear is severe. . . . At from 60 to 80 mm. . . . very severe. . . . Still higher pres-

sures produce agonizing pain. . . . At between 100 and 500 mm. . . . the tympanic membrane ruptures." With this in mind, observations were made of the altitudes at which onset of symptoms occurred in 46 cases of aero-otitis media.

Analysis of the scattergraph (chart 6) reveals that in all but 1 case the onset of symptoms occurred at 5,000 feet (1,500 meters) or above in the pressure chamber. In all but 5 cases symptoms developed at an altitude of 10,000 feet (3,000 meters) or above. The highest altitude at which the first symptoms appeared was 28,000 feet (8,500 meters); the lowest was 4,000 feet (1,200 meters). The average altitude at which symptoms appeared was 16,522 feet (about 5,000 meters).

In a chamber "flight" starting from 35,000 feet (10,600 meters), the subjects in whom aero-otitis media developed at an altitude of 16,522 feet underwent an average barometric pressure differential of approximately 224.8 mm. of mercury. In a "flight" from 30,000 feet (9,000 meters), as in the "cold run" and the "free fall," they underwent an average pressure differential of approximately 177.9 mm. of mercury. Any preventive measures aimed at reducing the incidence of aero-otitis media should be instituted, therefore, at altitudes above that at which symptoms develop in most subjects.

SUMMARY

Observations were made on the incidence of aero-otitis media in 167 subjects participating in 330 "high altitude flights" in a pressure chamber.

The incidence of symptoms plus objective otoscopic findings of aero-otitis media was 16.6 per cent, while the total incidence of objective otoscopic findings in the tympanic membrane and the middle ear was 27.9 per cent.

Observations in three types of "chamber flights" indicate that low temperature does not increase the incidence of areo-otitis media but that rate of descent and altitude attained are important factors.

A higher incidence of aero-otitis media was observed in subjects with obstruction of the eustachian tubes due to hypertrophied lymphoid tissue than in those with normal-looking ostiums.

The incidence of aero-otitis media was no greater in subjects with subacute infections of the upper respiratory tract than in subjects with no signs or symptoms of infection of the upper respiratory tract.

In this study the incidence of aero-otitis media was about the same in experienced flying personnel as in subjects with no flying experience.

Observations on 46 subjects showed that in the majority the symptoms of aero-otitis media appeared at about 16,000 feet.

Irradiation Clinic, 112th A. A. F. Base Unit, Westover, Mass.

Case Reports

FOREIGN BODY INVOLVING THE MAXILLARY ANTRUM

GEORGE A. RICHARDSON, M.D., PHILADELPHIA

A CASE of foreign body involving the antrum is presented because it illustrates that the persistence of a foreign body in a nasal accessory sinus always leads to chronic suppurative sinusitis and this, in turn, to suppuration elsewhere.

Schroder [1] stated that foreign bodies may enter through the nose or through openings in the antral walls caused by injuries or by dental trauma. The latter may be voluntary, surgical or accidental in extraction of a tooth or treatment of dental roots.

He stated further that the behavior of these foreign bodies in the sinus differs. Healing like that observed where foreign bodies are lodged in muscle and connective tissue does not occur. The most probable defense reaction would consist in an effort to expel the foreign thing. The literature contains reports of cases in which small foreign bodies have been expelled through the maxillary ostium, with more or less marked inflammatory symptoms. The mere presence of the foreign body may prove mechanically disturbing. It may lead to increase of mucosal secretion and interfere with outflow of the secretion. There is, furthermore, the danger of latent infection. On and about foreign bodies one may expect staphylococci, influenza bacilli, meningococci, colon bacteria, the bacilli of malignant edema, gas-forming bacilli and tetanus spores. Any day, something or other may lead to mobilization of the organisms with the result that they are disseminated through the body, and formation of abscesses, gangrene and tetanus may follow. If the infection extends to the orbit and venous thrombosis occurs, the sphenoid sinus may become involved with life-threatening complications.

Schroder also stated that, owing to the deposits of bone and metal splinters, which constitute infected foreign bodies, initially seromucous discharge is transformed to suppuration of a chronic type. The purulent inflammations are often fetid.

"In the majority of cases, however," said Schroder, "suppuration ensues and discharge and inflammatory edema persist as long as the foreign bodies remain in the sinus."

In some cases a foreign body remaining in the sinus for a long period is completely covered by inorganic salts deposited from secretion.

Most foreign bodies can be easily removed by the Caldwell-Luc operation, according to Schroder.

Another case of a pin in the maxillary antrum has been reported.[2]

From Otolaryngological Service B, Protestant Episcopal Hospital.

1. Schroder, G. M.: Fremdkörper in der Kieferhöhle, Wurzburg, 1937.
2. Canadian M. A. J. ——: ——, 1933.

REPORT OF A CASE

A woman 69 years of age was examined Jan. 11, 1944. At the age of 10 years she had an upper left premolar tooth extracted. Noticing a blood clot in the socket, she tried to dislodge it with a pin. The pin was lost in the antrum and evidently forgotten for a period of fifty-nine years.

At examination this patient stated that she had had nasal discharge and coughing off and on for the past fifty years. During the last few years the sputum had been blood tinged.

The nose revealed purulent discharge, especially in the area of the left ethmoid sinus, with granulation tissue.

The roentgen report was as follows: "There is gross disease of the paranasal sinuses. The left antrum is dense as if there was gross thickening of its mucosal lining with presence of thick exudate.

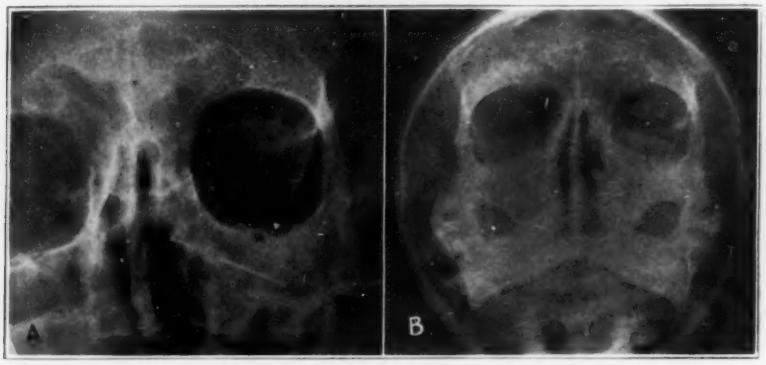

Fig. 1.—*A*, roentgenogram showing a pin in the left antrum. *B*, roentgenogram showing the left antrum clear.

Fig. 2.—Pin after removal. Owing to corrosion it broke into three pieces.

"There is a common pin apparently situated in the approximate position of the ostium of the left maxillary antrum. There is considerable disease of the right antrum but it is less than that present in the left antrum. There is an appearance of thickened membrane and possible exudate. There is similar disease of the frontal sinuses."

Figure 1 *A* shows the pin in the left maxillary antrum, and *B* shows the left maxillary antrum clear after removal of the pin.

The pin as removed is shown in figure 2.

On January 13, with the patient under local anesthesia and the influence of 3 grains (0.19 Gm.) of sodium amytal, intranasal antrotomy was done on the left side. A vertical incision 2 cm. in length was made anterior to the inferior turbinate and over the cristipyriformis. The cristipyriformis was removed with part of the wall of the canine fossa and 2 cm. of nasoantral wall under the inferior

turbinate. The antrum could be well visualized with this exposure, but it was not possible to see the pin.

A horizontal incision (Denker's operation) was then made. Above the margin of the gum, extending backward 4 cm., the pin was found, lying horizontal in the antrum as shown in figure 1 *A*.

Owing to corrosion, the pin broke in three pieces on its removal. The mucous membrane was not removed, because of the patient's age and condition and because of minimal gross pathologic change. The antrum was packed with one-half inch iodoform gauze, and the wound was closed. The patient made an uneventful recovery.

This patient had complicating pansinusitis and chronic bronchitis; otherwise the physical examination revealed no abnormalities.

I believe it is the duty of the surgeon to remove a foreign body as soon as possible to prevent possible disastrous consequences.

1532 Overington Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

ADVANCES IN THE UNDERSTANDING OF RHINOLOGIC AND OTOLOGIC CONDITIONS RELATED TO THE NERVOUS SYSTEM

A Critical Survey of Recent Literature

WELLS P. EAGLETON, M.D., Sc.D.
NEWARK, N. J.

(Continued from page 189)

TINNITUS AND DEAFNESS IN CENTRAL LESIONS OF THE BRAIN

Deafness from Lesions of the Pons.

Anatomy of the Pons.—From a diagnostic standpoint the pons may be divided dorsoventrally into three portions:

1. The tegmentum,[92] lying in the center. It is the dorsal part of the pedunculus cerebri (the crus cerebri). It is also called area tegmentatis Forel—the diencephalomesencephalic junction. The tegmentum is associated with primitive kinetic and static mechanisms.

2. The basis, situated ventrally. The syndrome of disease of the pontile basis is predominantly motor, since the pyramidal tracts are located ventrally in the basis.

3. The tectum, situated dosally. It is the tectum mesencephali, the corpus quadrigeminum, which forms the roof of the midbrain. The superior colliculus [93] makes up the rostral part of the tectum (Krieg [94]).

Lesions of the Pontile Tegmentum.—A lesion limited to the central portion of the tegmentum on one side results in myoclonus of the palate, the corner of the mouth and other ipsilateral parts.[95] Palatal myoclonus may also develop after lesions of the ipsilateral dentate nucleus, with

92. The tegmentum (Latin, meaning cover) is the dorsal part of the cerebral peduncle (crus-cerebri) containing fibers to the cortex plus a portion of the pons—the dorsal portion of the pons, pars dorsalis pontis.

93. "The colliculus (mount) superior, originally an end station for vision in man, acts principally as a reflex centre for many impulses. It is concerned chiefly with light impulses" (Riley, H. A.: Atlas of the Basal Ganglia, Brain Stem and Spinal Cord Based on Myelin Stained Material, Baltimore, Williams & Wilkins Company, 1943, p. 541.

94. Krieg,[51] p. 185.

95. Freeman, W.: Palatal Myoclonus: Report of Two Cases with Necropsy, Arch. Neurol. & Psychiat. **29**:742-755 (April) 1933.

crossed olivary degeneration (case described by Freeman, Ammerman and Stanley [96]).

When paralysis of associated lateral movements of the eyes with preservation of convergence occurs, there is likelihood that the longitudinal fasciculus basilaris pontis is involved.[97]

The interpretation is that the pathways for lateral gaze lie in the posterior longitudinal fasciculus, running down in the lateral portion of the tract as far as the nucleus of the abducens nerve.

In only a few cases has the aforementioned syndrome been reported to result from an extrapontile lesion (abscess),[98] and in these cases the lesion was usually massive and was so placed as to exercise considerable pressure on the pons.

Lesions of the caudal portion of the pontile tegmentum involve the abducens and facial nerves either at their nuclei or in the intracerebral course of their roots. Thus, the sixth and the seventh nerve are usually affected together, and in some cases the eighth nerve also.

The medial lemniscus is liable to damage if there is an extensive lesion of the pontile tegmentum. Consequently, there is likely to be some defect in vibratory and position sense in the contralateral limbs, with a degree of incoordination superficially resembling that seen when cerebellar disorders are present.

Freeman stated that with a pontile lesion there is "loss of *hearing in one ear, usually the contralateral, from destruction of the lateral lemniscus* (lateral acoustic fillet) which carries the acoustic fibers upward —Krieg [99]; although a lesion of the *caudal portion of the tegmentum of the pons* will extend to the *auditory nuclei and their entering fibers.*" [Italics mine.—W. P. E.]

Reduction of Hearing from Damage of the Auditory Centers or Their Tracts.—If the lesion is in the caudal part of the pons, the loss of hearing is ipsilateral; if the lesion is in the cephalic part, the greater loss is contralateral. When the lesion occurs at a higher level, the deafness may be more severe in the contralateral ear, owing to involvement of the lateral fillet.

Lesions of the Pontile Basis.—Bilateral lesions of the basis of the pons are a frequent cause of pseudobulbar palsy (Pontoppidan [100]).

96. Freeman, W.; Ammerman, H. H., and Stanley, M.: Syndromes of the Pontile Tegmentum, Foville's Syndrome: Report of Three Cases, Arch. Neurol. & Psychiat. **50**:462-471 (Oct.) 1943.

97. Riley,[93] p. 572.

98. Bucy, P. C., and Weaver, T. A., Jr.: Paralysis of Conjugate Lateral Movement of the Eyes in Association with Cerebellar Abscess, Arch. Surg. **42**: 839-849 (May) 1941.

99. Krieg,[51] p. 147.

100. Pontoppidan, K.: Involuntaere og inadaekvate emotionelle udbrud ved lidelser i pons varolii, Hospitalstid. **4**:405-410, 1886. Foix, C., and Hillemand, P.: Contribution à l'étude des ramollissements protubérantiels, Rev. de méd., Paris **43**:287-305, 1926.

Lesions of the Pontile Tectum.—Such lesions may result in a major cerebellar syndrome because of interruption of the dentatorubral connections.[101]

Syndrome of Lesion of the Tegmentum with Interruption of the Median Longitudinal Fasciculus (Freeman,[102] Foville [103] and Féréol [104]) Causing Paralysis of the Associated Lateral Movements of the Eyes with Preservation of Convergence.—In addition to the conjugate deviation there is paralysis of the sixth, the seventh and sometimes the eighth or the fifth nerve of the same side.

Auditory and Vestibular Signs from Tegmental Lesions.—Freeman [96] reports 3 cases. In case 1 the patient had a deaf left ear, as the sixth, seventh and eighth nerves did not function on the left side. The case is reported as one of an acute attack of multiple sclerosis, with neurologic symptoms which "might be found in a case of hemisection of the pons extending in a cephalocaudal direction from the fifth to the eighth nucleus" (Freeman).

[No vestibular tuning tests were done. W. P. E.]

In case 3 there was a "hemorrhagic lesion of the pons apparently centered about the tegmentum of the left side in the neighborhood of the eminentia teres; [the hemorrhage] presumably ruptured through the floor of the fourth ventricle, completely destroying one medial longitudinal fasciculus, . . . the nucleus of the sixth nerve and the fibers of the seventh nerve on the left side, and probably some of the cochlear fibers as well."

Involvement of the Eighth Nerve.—"Hearing was diminished on the left side, and in the Weber test sounds were referred to the right. Douching of the left ear did not produce nystagmus, but douching of the right ear provoked lateral nystagmus of the right eye when the patient looked toward the right. Shifting of the position of the head produced pronounced vertical nystagmus in both eyes."

Disturbance of Hearing in Occlusion of the Superior Cerebellar Artery.

Although the syndrome of the occlusion of the superior cerebellar artery is well recognized clinically, Freeman and Jaffe [101] could find only eight reports in the literature. This represents a total of 22 cases since the first description of the syndrome by Mills [105] in 1908.

---

101. Freeman, W., and Jaffe, D.: Occlusion of the Superior Cerebellar Artery, Arch. Neurol. & Psychiat. **46**:115-126 (July) 1941.

102. Freeman, W.: Paralysis of Associated Lateral Movements of the Eyes, Arch. Neurol. & Psychiat. **7**:454-487 (April) 1922.

103. Foville: Note sure une paralysie peu connue de certains muscles de l'oeil, et sa liasion avec quelques points de l'anatomie et la physiologie de la protubérance annulaire, Gaz. hebd. de méd. **6**:146-150, 1859.

104. Féréol: Phthisie pulmonaire. Hemiplégie alterne incomplète. Paralysie de la sixième paire gauche; inaction conjugée du muscle droit interne de l'oeil droit; tubercle confluent de la protubérance annulaire, Union méd., Paris **15**:597, 1873.

105. Mills, C. K.: Hemianesthesia to Pain and Temperature and Loss of Emotional Expression on the Right Side with Ataxia of the Upper Limb on the Left, J. Nerv. & Ment. Dis. **35**:331, 1908. Spiller, W. G.: Remarks on the Central Representative of Sensation, ibid. **42**:399-418, 1915. (Pathologic report on Mills's case.)

The superior cerebellar branches of the basilar artery pass laterally at the upper border of the pons directly below the oculomotor nerve of the same side, turn around the lateral side of the pedunculus cerebri below the trochlear nerve and reach the upper surface of the cerebellum. They divide into (1) a medial branch, which supplies the vermis and the anterior medullary velum, and (2) a lateral branch, distributed over the upper surface of the lateral lobe, which anastomoses with the inferior cerebellar artery.[106]

Contralateral Deafness.—Contralateral deafness was observed by Mills, the lesion in his case involving the lateral lemniscus, which explains this symptom, although local causes were not ruled out. In the case observed by Freeman and Jaffe [101] the hearing was somewhat impaired ipsilaterally, possibly as a result of infarction in a region near the cochlear nucleus.

In the case of Freeman and Jaffe, at autopsy the left lateral lemniscus was followed upward through the brain stem to the inferior colliculus and the medial geniculate body of the same side. "At the level of the inferior colliculi and higher in the midbrain, no evidence of degeneration is seen."

This case is the first recorded one of occlusion of the superior cerebellar artery caused by an embolus in a patient with subacute bacterial endocarditis. Death occurred about two months after the apparent onset of the intracranial complication.

The patient, aged 44, had had "rheumatism" for ten years and during the past four years had suffered pains in the joints of the legs. For the past six months he had been in bed almost constantly because of pains in the knees and the ankles. On admission he complained of numbness in both legs; the left leg felt hot and the right cold.

His temperature was 101 F.; the pulse rate, 100; the blood pressure, 140 systolic and 50 diastolic. He was confused and dyspneic. The pupils were pinpoint type and failed to react to light or in accommodation. There were marked carotid pulsations, and the "entire bed shook with each cardiac systole." To and fro murmurs were heard over the aortic and mitral areas and were transmitted along the vessels of the neck.

The electrocardiogrm showed left axis deviation and delayed auriculoventricular conduction (0.28 second).

Petechiae were present in the mucous membranes, and blood cultures yielded organisms on three occasions; on two, nonhemolytic, and on one, hemolytic streptococci.

The red cell count was 3,000,000; the hemoglobin concentration, 50 per cent; the white cell count, 20,000, with 50 per cent segmented polymorphonuclears, 22 per cent band forms, 21 per cent lymphocytes and 7 per cent monocytes.

Urinalysis showed albumin, casts, occasional red blood cells and leukocytes.

An intracranial complication developed one week after admission: The patient had marked tremors of the left hand and arm. Neurologic examination showed:

1. Unequal pupils, the right being larger than the left; both reacted sluggishly to light.

2. A Horner syndrome on the left, with ptosis of the left lid, exophthalmos, miosis of the left pupil and absence of sweating on the left side of the face.

---

106. Cunningham, D. J.: Textbook of Anatomy, ed. 5, New York, William Wood & Company, 1919, pp. 907-908.

3. Slight amimia (loss of power to express ideas by signs) of the left side of the face. This appeared on smiling and marked paresis of the right side of the face on wincing, but no facial weakness was noted on voluntary motion.

4. Bilateral impairment of hearing: On the right, a watch was not heard at all; in the left, it was heard at 2 inches (5 cm.)—normally it should be heard at 10 inches (25.4 cm.). The Weber test showed lateralization to the left (the better hearing ear); bone conduction was greater than air conduction (whether in one ear or both is not stated).

5. Cerebellar signs: hypotonia involving the upper and lower extremities on the left side, rebound phenomenon, intention tremor, dysmetria, asynergia and adiadokokinesis. Speech was thick and difficult to understand.

6. Analgesia to temperature, pinprick and pain from deep pressure over the right side of the body, including the face, and hypesthesia to light touch.

7. Increased deep tendon reflexes, more active on the right; a positive Hoffmann and an equivocal Babinski sign on the right. The abdominal and cremasteric reflexes were absent.

8. Slight nuchal rigidity and bilateral Kernig sign.

At necropsy the left cerebellar hemisphere appeared on palpation to be somewhat softened at the lateral margin of the anterior lobule. Removal of the arachnoid which formed the cisterna pontis revealed the superior cerebellar arteries. The left superior cerebellar artery was yellowish, narrow, tortuous and nodular; it was completely occluded for about 2.5 cm. of its length, from its origin at the basilar artery to its visible termination.

After fixation of the brain, an area of brownish discoloration was seen on the superior surface of the left cerebellar hemisphere, involving the lateral half of the dentate nucleus, and extending into the brachium conjunctivum and the dorsolateral tegmentum of the pons.[107]

Cerebral Tinnitus.

The Diagnostic Importance of (1) *Inability to Mask, and* (2) *the Augmenting of the Tinnitus into Greater Prominence by Masking.*—Fowler[108] states that there are some forms of "nonvibratory tinnitus" that are difficult or impossible to mask even with the loudest sound (the loudest thermal noise) in spite of the fact that the loudness of the tinnitus, measured audiometrically, may be not over 5 or 10 decibels. This type of tinnitus often sounds just like a thermal noise.

---

107. Critchley, M., and Schuster, P.: Beiträge zur Anatomie und Pathologie der Arteria cerebelli superior, Ztschr. f. d. ges. Neurol. u. Psychiat. **144**:681-741, 1933. Williams, T. J.: Partial Thrombosis Affecting Supply of Left Inferior Cerebellar Artery: Dysphagia, J. Laryng. & Otol. **45**:883 (Dec.) 1930; abstracted, Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **26**:534-582 (Nov.); 690-735 (Dec.) 1937. Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1941, reprint (1937), p. 33.

108. Fowler, E. P.: Head Noises in Normal and in Disordered Ears: Significance, Measurement, Differentiation and Treatment, Arch. Otolaryng. **39**:498-503 (June) 1944.

Another phenomenon is that in some cases the masking noise seems to bring the tinnitus into more prominence, the masking noise making the tinnitus appear louder.

In order to account for the phenomena of difficult masking, Fowler offers the hypothesis that "this type of tinnitus (i. e. difficult to mask) arises at least in part in the central neurons or the central nuclei." He sees no reason why nonvibratory tinnitus cannot be set up in the central neurons, because a spatial arrangement of the auditory nerve fibers and nuclei is maintained in the tuberculum acusticum—the dorsal nucleus of the cochlear nerve situated on the corpus restiforme—and even in the higher levels (the mesial geniculate ganglions). Thus a lesion anywhere along these cerebral pathways may set up disturbances in a manner similar to that observed in disturbances set up in other sensory tracts. He states: "Herein lies almost a virgin field for research."

Comment (W. P. E.).—Central tinnitus is a perception phenomenon allied to hearing much as pain is allied to sensation. It is not a pure electrostate as is hearing. This is the reason, in my opinion, why it cannot be masked by other *sounds,* either thermal or tonal; although the nature of the primary auditory cells in the dorsal cochlear nuclei (tuberculum), which are different from the secondary auditory cells, may allow the production of tinnitus.[109]

Central Disturbances of Hearing.

Fowler [110] observed "2 patients with monaural total, or almost total, acquired inability to recognize words or language, although the speech frequencies, the cadence and the variations in intensity of articulate speech were sensed. There was not sufficient audiometric loss in the speech frequencies to account for it. Sounds heard were useless as an aid to hearing speech. A hearing aid was of no use. In the opposite ear hearing was excellent." Analysis and correlation of his audiometric and anatomic observations lead him to the conclusion that "the syndrome described, i. e., monaural deafness for speech without sufficient loss of hearing for the speech frequencies to account for it and without a loss of ability to remember or to produce speech properly, and usually with a persistent tinnitus difficult to mask (and may be a transitory vertigo), may in part at least be due to lesions central to the end organ—in its primary nuclei or somewhere in the central auditory neurons to the same or the opposite side of the brain (or both), depending on whether the symptoms are in the ear homolateral or [the ear] contralateral to the dominant area of the cortex."

---

109. Krieg,[51] p. 145, fig. 97; p. 148, fig. 100.

110. Fowler, E. P.: Total Monaural Deafness for Speech But Not for Speech Frequencies: Misleading Threshold Audiograms, Arch. Otolaryng. **41**:377 (May) 1945.

"Pathways from the cochlea and through the primary acoustic nuclei go to both sides of the brain. There are several crossings in the medulla, and fibers from the two ears join each other at the level of the geniculate body and go together to the cortex. If there were a lesion in the geniculate body or the cortex, it would be difficult to understand how one ear hears well while the other one is not capable of analyzing speech. Therefore by exclusion one has to place the lesion in the cochlea or in the primary nuclei of the affected side" (Lorente de Nó).

Advances in Understanding of Word Deafness in Abscess of the Pia-Arachnoid (Nielsen).

Word deafness is "perception agnosia" (absence of the faculty of perceiving the form and the nature of spoken words). It occurs in intracranial suppuration of otitic origin, in which case it is pathognomonic of extensive abscess of the pia-arachnoid that has extended upward from the region of the tegmen tympani,[111] involving the posterior part of Brodmann's area 22 and the upper part of area 39. This area Nielsen [112] calls "the language formation area"—because language is learned by association of vision and hearing with objects and symbols.

Word deafness occurring with an abscess is always transient.

It may occur with abscess and with hemorrhage of the "the language formation area" but never with tumor, because the opposite cortex, of the nondominant hemisphere, soon takes on the function. This is because hearing pure and simple is bilaterally placed and word deafness is an association deafness, a combination of (*a*) hearing and (*b*) proper perception of words.

Wernicke's area [113] (Brodmann's areas 41 and 42) is a crescentic zone in the posterior third of the superior temporal convolution just lateral to the transverse gyri of Heschl.[114] It is the "secondary auditory area" concerned with the two functions of (*a*) recognition and (*b*) reauditorization of sounds in general, with exclusion of music.

Music Area.—The area for recognition of music is anterior to Wernicke's crescentic zone in the anterior half of the superior temporal convolution. For this reason a patient with a lesion of the posterior portion of the superior temporal convolution may fail to comprehend spoken language and still recognize and appreciate music (Nielsen [115]).

---

111. Eagleton,[21] p. 179 (see case VII, J. C. C., and also postmortem report on p. 35).

112. (*a*) Nielsen, J. M.: A Text-Book of Clinical Neurology, New York, Paul B. Hoeber, Inc., 1941. (*b*) Nielsen, J. M., and Fitzgibbon, J. P.: Agnosia, Apraxia, Aphasia, Los Angeles, The Los Angeles Neurological Society, 1936.

113. Wernicke's sensory speech center (Karl Wernicke, 1848-1905) for the understanding of the spoken word, located in the first temporal convolution and in the supramarginal gyrus. Wernicke's area comprises the supramargin and angular gyri and the bases of the first and second temporal convolutions, where Marie locates the speech center (Stedman, T. L., and Garber, S. T.: Stedman's Practical Medical Dictionary, ed. 14, Baltimore, William Wood & Company, 1939, p. 1230).

114. Krieg,[51] p. 318, fig. 197, which shows areas of the lateral aspect of the human cerebral cortex (after Brodmann).

115. Nielsen,[112] p. 240.

Comment (W. P. E.).—In the examination of considerably over 100 patients with abscess of the temporosphenoidal lobe I have encountered word deafness but twice. In both cases the suppuration was in the subdural space (Eagleton [116]). On the other hand, naming aphasia is frequent in patients with abscess occurring within the temporal sphenoidal lobe.

AUDITORY AND OLFACTORY HALLUCINATIONS

Illusion versus Hallucination.

An illusion is a subjectively distorted perception of a real object; hallucination, a mental image projected outward in the absence of an external stimulus. However, hallucinations often make use of external objects as their starting point, and since no sense organ is ever entirely free from the stimuli of external objects, it is theoretically possible that every hallucination has a peripheral stimulus.[117]

Auditory and Olfactory Hallucinations in General Diseases.

In auditory hallucinations the noises seem to the patient (*a*) to be outside himself and (*b*) to have a significance which ordinary tinnitus does not possess. They are real noises.

Hallucinations have been observed more frequently in the course of dementia paralytica since the advent of malarial therapy.

"Stransky's symptom of thought-echo, reading-echo, or *Gedankenhören* belongs as a rule to the domain of psychiatric medicine, though it has been known to occur in patients with temporal lesions (Sanz; Klein). It may show itself as an auditory hallucination whereby the patient hears his own thoughts spoken aloud into his ear" (Critchley).

116. Cases of abscess of the brain with word deafness have been reported by: Brown, R. G.: Mastoid Disease with Cholesteatoma Complicated by a Cerebral Abscess, M. J. Australia **1**:212 (March 6) 1920. Wiener, A.: A Case of Brain Abscess Following Traumatism and Acute Mastoiditis: Operation; Recovery, Arch. Otolaryng. **35**:340-346, 1906. Laignel-Lavastine: Aphasia et apraxie, Bull. méd., Paris **34**:277, 1920. Halphen, E.; Djiropoulos and Laignel-Lavastine: Aphasie de Wernicke par abcès cérébral d'origine otique, Rev. d'oto-neuro-ocul. **5**:299-302 (April) 1927; abstracted, Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1941, reprint (1928), p. 25. Watkyn-Thomas, F.: Localization of Cortical Centre for Hearing, J. Laryng. & Otol. **42**: 505 (Aug.); 593 (Sept.) 1927. Schuster, P., and Taterka, H.: Beitrag zur Anatomie und Klinik der reinen Worttaubheit, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:494-538, 1926; abstracted, Arch. Neurol. & Psychiat. **19**:153 (Jan.) 1928; abstracted, Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1941, reprint (1928), p. 26.

117. McCowan, P. K.: Significance of Auditory and Visual Hallucinations, Brit. M. J. **2**:631-634 (Sept. 23) 1939; abstracted, Neuro-Psychiatric Institute of Hartford Retreat, Nov. 11, 1939, series 7, no. 240.

In milder febrile illnesses, isolated hallucinations of hearing may occur, with no background of confusion and nothing incongruous about them.

Hallucinations may occur as auditory, visual or combined sensory auras in cases of essential epilepsy.

The simplest forms of auditory hallucinations are vague sounds and noises. Hallucinations of music and singing are comparatively rare except in confusional states, especially delirium tremens (McCowan [117]). General auditory hallucinations are characteristic of the chronic psychoses. Auditory hallucinations are a final confirmation of old fears or hopes.

Auditory hallucinations do not ordinarily occur in migraine. The presence of pulsatile tinnitus during an attack of migraine should arouse suspicion of an underlying vascular anomaly of the brain, for example, angioma (Critchley [118]).

Colored audition, according to Marinesco and Vasile,[119] is a psychologic condition, the auditory centers for words and for mental vision being involved.

Berggren [120] expressed the opinion that auditory hallucinations occur in a large percentage of insane persons with aural diseases. Courville [121] found auditory hallucinations in 4 of 99 patients. These symptoms were the result of irritation rather than of destruction of the auditory cortex. Damage of the auditopsychic area led to impaired understanding.[122]

Comment (W. P. E.).—The pathways of vision and audition are close together at (*a*) the colliculi, the superior and inferior eminences (quadrigeminal bodies), and (*b*) the geniculate bodies, although they are widely separated at their origins and at their terminations. This anatomic fact may help to explain intersensory connections and partic-

118. Critchley, M.: Neurological Aspect of Visual and Auditory Hallucinations, Brit. M. J. **2**:634-639 (Sept. 23) 1939; abstracted, Neuro-Psychiatric Institute of Hartford Retreat, Nov. 11, 1939, series 7, no. 243.

119. Marinesco, G., and Vasile, S.: Etudes sur l'audition colorée, Rev. d'oto-neuro-opht. **7**:500-503 (July) 1929; abstracted, Arch. Neurol. & Psychiat. **23**:818 (April) 1929.

120. Berggren, S.: Die Beziehungen zwischen Gehörhalluzenationen und Gehörorgan, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **120**:141-163 (March) 1929.

121. Courville, C. B.: Auditory Hallucinations, J. Nerv. & Ment. Dis. **67**:265-274 (March) 1928.

122. Lhermitte, J.; Delthil, and Garnier: Syndrome contralateral du noyau rouge avec hallucinations visuelles et auditives, Rev. neurol. **70**:623-628 (Dec.) 1938; abstracted, Neuro-Psychiatric Institute of Hartford Retreat, Feb. 17, 1939, series 7, no. 38. Brun, R.: Troubles de l'odorat et du gout comme signe d'une lesion focale du lobe temporal, J. belge de neurol. et de psychiat. **37**:262-269 (April) 1937; abstracted, Arch. Neurol. & Psychiat. **39**:1324-1325 (June) 1938.

ularly the symptom called synesthesia—"the experience of a sensation in one place due to stimulation applied to another place" (Eagleton [123]).

Auditory Hallucinations Caused by Encephalic Tumors.

The patients showing auditory hallucinations may be divided into three groups: (1) those with tumor of the frontal lobe, (2) those with tumor of the temporal lobe and (3) those with multiple tumors of the brain. Voices are associated with evidence of great increase in intracranial pressure, the phenomena probably resulting from trauma (Courville [121]). The posteroinferior third of the cerebral hemisphere seems to be of the greatest significance in tumoral hallucinations—that is to say, the regions closely associated with the end stations of the special sense pathways. When the tumor is situated more anteriorly to the occipital lobe, it may be associated with auditory phenomena. "I hear a sort of 'ha-ha'-ing in the ears" (Critchley [118]).

Auditory hallucinations (Critchley [118]) comprising crude noises such as buzzing or roaring occur with tumors of the temporal lobe. Auditory hallucinations alone do not ordinarily occur. In tumoral cases there are also auras of sudden loss of vision or hearing, implying perhaps negative visual or auditory hallucinations.

Infrequency of Auditory Hallucinations in Patients with Tumor of the Brain.—Cushing has remarked that in his experience auditory disturbances are conspicuous by their absence.

In Cushing's clinic, auditory hallucinations occurred four times among 99 patients with tumors of the temporal lobe and six times among 99 with tumors of the frontal lobe.

Kolodny noted an aura of "horrible noises" once only in 38 consecutive patients with temporal tumors.

Classification of Auditory Hallucinations Caused by Encephalic Tumors.—Tarachow [124] states that 96 of 458 patients with supratentorial tumor of the brain had hallucinations. Auditory hallucinations (formed and unformed) occurred in 46, olfactory hallucinations in 20, gustatory in 3, tactile in 6 and visual in 37.

Auditory Hallucinations: "Voices," "melodies" or "music other than bell sounds" were classed as "formed auditory hallucinations" and occurred in 6 patients. One patient with formed auditory hallucinations suffered from a lesion confined to the temporal lobe. When the patient described experiences such as grinding, roaring, buzzing, whistling, ringing, cracking or any monotonous meaningless noise, they were classed as unformed auditory hallucinations. Five patients had formed auditory hallucinations in combination with other types of hallucinations. Two of these had a tumor of the temporal lobe; one had a frontotemporal tumor; two had a tumor confined to the parietal lobe and one a diffuse lesion.

123. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology: Critical Survey of Recent Literature, Arch. Otolaryng. **32**:54-120 (July) 1940.

124. Tarachow, S.: The Clinical Value of Hallucinations in Localizing Brain Tumors, Am. J. Psychiat. **97**:1434-1442 (May) 1941.

Conclusions of Tarachow.—(1) The clearest localization was afforded by formed auditory hallucinations, which were primarily due to lesions of the temporal lobe, the temporal lobe being involved in 4 of the 6 patients with formed auditory hallucinations. (2) Involvements of the temporal lobe showed a high incidence of unformed auditory and olfactory hallucinations, while (3) the incidence of formed auditory and gustatory hallucinations was low. (4) There is a relatively high incidence of olfactory and unformed auditory hallucinations in lesions of the temporal lobe; the frontal and parietal lobes may also be involved. (5) Unformed auditory hallucinations were related predominantly to lesions of the frontal and temporal lobes, though all the lobes were involved, as well as midline structures, notably the pituitary gland (8 cases); such hallucinations occurred in a few cases of lesions of the pineal gland and the third ventricle. With lesions of the lobes the hallucinations tended to be heard on the side of the lesion. (6) Unformed auditory hallucinations were most commonly followed by unformed visual and then olfactory hallucinations. (7) Gustatory hallucinations tended to appear together with olfactory hallucinations and to be related to lesions of the temporal lobe.

"Dreamy State."—The psychosensory phenomena which may accompany discharging lesions of the uncinate lobe, the so-called "dreamy states," usually take the form of olfactory or olfactogustatory hallucinations but occasionally those of visual and auditory types.

Comment (W. P. E.).—I have seen auditory and visual hallucinations associated with a deep-seated abscess of the brain following a septic infarct. The hallucinations were interpreted by the consulting neurologist as of toxic origin (Eagleton [125]).

Hallucinations with Disturbances of the Equilibratory Apparatus.

Bonnier showed that disturbances of the equilibratory apparatus can cause curious modifications of the personality. False perceptions engendered by disorders of the vestibular apparatus have a special bearing on the consciousness the patient has of the body, "the image of the corporeal me."

In the cases reported by Lhermitte and Bineau [126] the first patient had hallucinations of the corporeal schema and visual hallucinations, accompanied by intense vertigo, nystagmus, vomiting and a sensation of spatial displacement. In the second patient the head seemed to lie to one side. In both patients a lesion of the central vestibular apparatus was evident, all symptoms being referable to the rhombencephalon.

125. Eagleton,[21] case 17, pp. 77-81.

126. Lhermitte, J., and Bineau: Hallucinations et fonctions vestibulaires, Rev. d'oto-neuro-opht. **17**:241-244 (April) 1939; abstracted, Arch. Neurol. & Psychiat. **43**:165-166 (Jan.) 1940.

LARYNGEAL EPILEPSY

Babbitt [127] reports a case of laryngeal epilepsy:

Chronic laryngitis followed an attack of grip. The hoarseness varied in degree, but the sore throat was present at all times, associated with stuffiness of the eustachian tubes and some pain in the right ear.

In the course of examination, a pillar retractor drew back the anterior pillar of the left side, and cryptic exudate was expressed from the upper pole of the tonsil. The patient suddenly became cyanotic, with tremor of the body, twitching of the hands and laryngeal spasm—a typical epileptiform spasm, lasting scarcely a minute. He had had two definite attacks of the same character prior to this, one occurring six years before and a second one following a crabmeat dinner three years previously.

Babbitt thinks that there were two contributing factors—(1) the marked nasal block of the cartilaginous septum and (2) the exquisite tenderness and the injection of the left tonsil.

Later, during removal of the tonsils, an abscess cavity was found posterior to and involving the left upper pole of the tonsil.

Whitty [128] states that in laryngeal epilepsy slight mental confusion is more frequent than true loss of consciousness or epileptiform movements. He believes that the neurogenic factor at play is a true reflex laryngeal epilepsy, with a laryngeal aura or venous congestion caused by violent coughing. It occurs in persons in whom the cerebral cortex is predisposed to epilepsy either from vascular degeneration or from other factors.

The Auditory Threshold During Attacks of Petit Mal.

Schwab [129] studied the difference of the auditory reaction time during petit mal attacks—viz., inability to execute purposeful movements from auditory stimuli (a compressed small steamboat air whistle) with the visual stimuli that arise from a neon light bulb. Auditory stimuli were found to have a greater terminating ability.

"The intensity of the neon light was measured with a Weston light meter, and at full brilliance (110 volts) gave a reading at 16" of 6.4."

It may be concluded that both light and sound stimuli tend to terminate moderately severe petit mal seizures, the former being much more effective (Gibbs and others [130]; Berger [131]).

---

127. Babbitt, J. A.: Laryngeal Epilepsy with Symptoms Conforming to the Pattern of Substantiated Cases Reported in the Literature, Tr. Am. Laryng. A. **62**:54-56, 1940. A bibliography of twenty similar experiences is attached.

128. Whitty, C. W. M.: The So-Called "Laryngeal Epilepsy," Brain **66**: 43-54 (March) 1943. He reports 4 cases.

129. Schwab, R. S.: The Influence of Visual and Auditory Stimulation on the Electroencephalographic Tracing of Petit Mal, Am. J. Psychiat. **97**:1301-1312 (May) 1941; abstracted, Arch. Neurol. & Psychiat. **46**:160 (July) 1941.

In discussion Dr. Herbert Jasper, of Montreal, Canada, pointed out the fact that petit mal discharges, as they were called, can be destroyed, depressed or inhibited by a loud sound is fundamental. This was mentioned previously by Dr. Lennox in recommending a stimulating regimen for epileptic patients.

It is assumed that the failure to respond to a stimulus and a subsequent statement of the patient that the stimulus was not observed indicate that the patient was in an abnormal state of unresponsiveness for which one may employ the term "unconsciousness."

Comment (W. P. E.): This observation may be of great importance in a better understanding of the psychology of hearing. It suggests that not only is (1) the threshold of "sound impulses" below that of visual impulses but also that (2) sound impulses are a more dominant influence in cases of "impairment of consciousness."

OTITIC ENCEPHALITIDES

Cases of encephalitis may be divided into three groups caused by: (*a*) bacteria, protozoa and other parasites, (*b*) viruses and (*c*) unknown agents (Dingle [132]).

Rosenow [133] sums up the relation of neurotropic streptococci to encephalitis and encephalitic virus by stating (1) that the streptococcus is a source of what is now considered virus, (2) that virus represents the filtrable phase of the streptococcus and (3) that in the epidemic of 1941 spread of streptococci by air was a major factor in causing encephalitis to occur over such a vast area.

Nonsuppurative Encephalitis Complicating Otitis Media.

Leegaard [134] reports 2 cases of otogenous encephalitis in which aphasia and hemiplegia developed.

In case 1 a woman 37 years old suffered from acute otitis and mastoiditis. Symptoms of severe encephalitis appeared two to three days after mastoidectomy was performed. Symptoms of meningitis strongly suggested a focal lesion in the left temporal fossa. Multiple punctures of the brain yielded negative results.

130. Gibbs, F. A.; Davis, H., and Lennox, W. G.: The Electro-Encephalogram in Epilepsy and in Conditions of Impaired Consciousness, Arch. Neurol. & Psychiat. **34**:1133-1148 (Dec.) 1935.

131. Berger, H.: Ueber das Elektroenkephalogramm des Menschen, Arch. f. Psychiat. **87**:527-570, 1929.

132. Dingle, J. H., cited by Reese, H. H.; Lewis, N. D. C., and Sevringhaus, E. L.: The 1943 Year Book of Neurology, Psychiatry and Endocrinology, Chicago, The Year Book Publishers, Inc., 1942, p. 118; Encephalitis of Virus Etiology, New England J. Med. **225**:1014-1022 (Dec. 25) 1941.

133. Rosenow, E. C.: Relation of Neurotropic Streptococci to Encephalitis and Encephalitic Virus, Proc. Staff Meet., Mayo Clin. **17**:551-560 (Nov. 4) 1942.

134. Leegaard, T.: On Otogenous Encephalitis, Acta oto-laryng. **27**:46-54, 1939.

Despite the increased number of cells in the spinal fluid, the clinical symptoms of meningitis were not pronounced. After four days the spinal fluid was normal. For this reason Leegaard thinks that a bacterial infection of the meninges could not have occurred.

Borries [135] in 1921 classified cases of otitic encephalitis into (1) those of: encephalitis simplex (without distinct clinical symptoms) and (2) those of encephalitis hemorrhagica nonsuppurativa. He held that encephalitis may be a hematogenous or a lymphogenous infection; the larger number of lymphogenous infections develop in the immediate neighborhood of the primary focus (Korner's law), but many appear in the vicinity of the artery of the sylvian fissure (Oppenheim).

Pathologic Aspects of Otitic Encephalitis.—Borries claimed to have found beneath the pia mater at operation a hemorrhagic zone with a corresponding softened zone. Microscopic examination revealed dilatation of the capillary and lymphatic vessels with perivascular leukocytic and red cell infiltration and softening from degeneration of the nerve tissue.

Comment (W. P. E.): There are no lymphatic vessels in the brain. Borries evidently was referring to perivascular arachnoid spaces (Virchow-Robin spaces).

Nonsuppurative Encephalitis with Jacksonian Epileptic Seizures from Otitis Media.—Smith [136] observed a girl aged 11 whose ears discharged for sixteen days and then ceased. Seven days later she lapsed into a stuporous state and had a convulsion. The later Jacksonian convulsions consisted of clonic movements of the left arm, then of the left side of the face and last of the left leg.

She maintained a left-sided cerebellar attitude, to which she reverted regularly.

The neurologist noted paresis of the left side of the face, weakness of the left upper extremity, a bilateral Babinski sign, absence of all deep reflexes and abdominal reflexes and blurring of the optic disks on the nasal side.

The spinal fluid was colorless, under normal pressure, with globulin and sugar contents normal.

The neurologist's impression was that "she possibly had a right temporosphenoidal abscess, causing upward pressure on the anterior central gyrus, irritating the face and arm centers."

The roentgenologist reported cloudiness over the left mastoid process and destruction of the cells in the upper part of the right mastoid process.

Simple bilateral mastoidectomy revealed necrosis throughout the mastoid processes, including the inner table.

Two days after the operation the patient came out of coma. On the third postoperative day speech returned. The patient left the hospital on the ninth postoperative day.

---

135. Borries, G. V. T.: Otogenous Encephalitis, Hospitalstid. **64**:545, 1921; Ztschr. f. d. ges. Neurol. u. Psychiat. **70**:93, 1921.

136. Smith, C. H.: Otogenous Encephalitis, Laryngoscope **53**:392-396 (June) 1943.

Review of Literature: Oppenheim in 1900 [137a] and Voss [137b] in 1902 reported cases of "nonpurulent otogenous encephalitis." Adson [138] in 1924 named it "pseudobrain abscess." Yaskin,[139] in a study of 326 patients with neurologic complications of aural and nasal infections, found 4 having "localized nonsuppurative encephalitis."

In advising against exploration Smith quotes Eagleton [140]: "Any one who has microscopically examined the cerebral tissue along the track of a diagnostic puncture must appreciate the falsity of [the statement that puncture is harmless]. Any puncture is attended with some danger, especially in the presence of a suppurative process in the neighborhood."

Encephalitis After Infection of the Upper Respiratory Tract.

In 8 cases of encephalitis Adler [141] noted a history of "colds" or infection of the frontal or the ethmoid sinuses from four to twelve days before the onset of neurologic signs. In 5 cases there was involvement of the optic and oculomotor nerves. The spinal fluids showed lymphocytes as the predominant cells.

Encephalitis After Otitis Media or Mastoiditis.—The pathologic picture is that of localized encephalitis extending from a meningeal exudate or abscess into the brain substance. Meningeal signs dominate the clinical picture.

Encephalitis Associated with Otitis in Infants.—Associated with otitis media or mastoiditis there may occur encephalitis characterized chiefly by hyperemia and new formation of capillaries. It belongs to the group of postinfectious toxic encephalitides (Grinker [142]). In reported cases, mostly among children,[143] the spinal fluid findings were identical with those in cases of abscess of the brain. However, no pus could be found on exploration, and the patients recovered.

---

137. (*a*) Oppenheim, H.: Zur Encephalitis acuta non purulenta, Berl. klin. Wchnschr. **37**:201-204, 1900. (*b*) Voss, O.: Otitis media und Encephalitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:596-608 (May 10) 1928.

138. Adson, A. W.: Pseudobrain Abscess, S. Clin. North America **4**:503-512 (April) 1924.

139. Yaskin, J. C.: Neurologic Complications of Infections of Temporal Bone and Paranasal Sinuses, Arch. Otolaryng. **30**:360-388 (Sept.) 1939.

140. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **10**:636-686 (Dec.) 1929.

141. Adler, A.: One Hundred Cases of a Condition Diagnosed as Acute Encephalitis: A Clinicopathologic Study, Arch. Neurol. & Psychiat. **44**:541-567 (Sept.) 1940.

142. Grinker R. R.: Neurology, Springfield, Ill., Charles C Thomas, Publisher, 1937.

143. Symonds, C. P.: Localized Non-Suppurative Encephalitis, Proc. Roy. Soc. Med. (Sect. Otol.) **20**:1142 (May) 1927. Atkinson, M.: Localized Nonsuppurative Encephalitis Adjacent to Focus of Infection in the Skull, Arch. Neurol. & Psychiat. **41**:556-564 (March) 1939. Adson.[138]

An infant aged 1 year had generalized convulsions, with both ears discharging. After the convulsions had subsided there was right hemiplegia. The spinal fluid, taken sixteen days after the onset of convulsions, was clear and contained 40 cells per cubic centimeter, chiefly polymorphonuclear leukocytes; the total protein content was 24 mg. and the sugar content 40 mg. per hundred cubic centimeters.

Factors in the Production of Otorhinogenic Hydrocephalus.

In a discussion of otorhinogenic hydrocephalus Reeves [144] suggests that the factors in its production are (1) interference in the absorption of spinal fluid due to previous inflammatory lesions of the pacchionian bodies or the perineural prolongations of the arachnoid, (2) hypersecretion of spinal fluid, (3) closure of the interventricular foramen or of the foramens of Luschka and Magendie or adhesions producing closure of the cisternas and (4) hypertension of the cerebrospinal fluid, which may occur if the sigmoid sinuses and the jugular foramens are asymmetric and the side of the larger one is involved, a condition which could slow down the venous circulation.

(1) *Davidoff and Dyke's Sign of Otitic Hydrocephalitis, Pseudoabscess of the Brain and Serous Meningitis;* (2) *Sitting Position in the Treatment of Increased Intracranial Pressure.*

"Otitic hydrocephalus," [145] "serous meningitis," "pseudotumor of the brain," "pseudoabscess of the brain," "intracranial serous effusions" and "intracranial hypertension of unknown cause" [146] are terms applied to a specific syndrome of increased pressure of the cerebrospinal fluid marked by (*a*) headache, (*b*) failing vision, (*c*) papilledema and (*d*) occasionally vomiting.

The impression of (1) "apparent general well-being of the patient and normal conditions (except for papilledema)" is stressed by Ecker, Ayer and O'Connor,[147] as is the fact that (2) the ventricles of the brain are "normal in size, shape and position." To these normal signs is now to be added (3) Davidoff and Dyke's [148] sign of *immediate extrusion of air into the subdural space.*

Davidoff-Dyke Sign of Increased Pressure in Serous Meningitis.—Davidoff and Dyke regard their sign as of diagnostic value in serous

144. Reeves, E.: Otorhinogenic Hydrocephalus, Arch. Otolaryng. **34**:920-935 (Nov.) 1941.

145. Williams, H. L.: Otitic Hydrocephalus, Arch. Otolaryng. **25**:632-652 (June) 1937.

146. Sahs, A. L., and Hyndman, C. R.: Intracranial Hypertension of Unknown Cause: Cerebral Edema, Arch. Surg. **38**:428-442 (March) 1939.

147. Ecker, A. D.; Ayer, W. D., and O'Connor, F. J.: Increased Intracranial Pressure Attributed to Venous Obstruction: Beneficial Effect of Upright Position, J. A. M. A. **114**:1440-1442 (April 13) 1940.

148. Davidoff, L. M., and Dyke, C. G.: Series of Cases of Serous Meningitis, Arch. Neurol. & Psychiat. **36**:1378-1380 (Dec.) 1936.

meningitis. It consists of "extrusion of the air from the ventricles out to the subdural space under the burr holes during ventriculography."

Sitting Position in the Treatment of Continued Increased Intracranial Pressure Following Operative Obliteration of the Lateral Sinus.—In Ecker's[147] case normal pressure of the cerebrospinal fluid was accompanied by persistently increased intracranial tension and choked disks apparently the result of packing of the lateral sinus.

The increased intracranial pressure responded neither to rigorous dehydrating measures nor to ventricular punctures. Maintenance of the patient's head higher than the rest of the body was followed by prompt disappearance of intracranial hypertension and early subsidence of the choked disks; the posture apparently enabled the venous blood to drain through the remaining and collateral channels.

The authors suggest that sterile thrombosis of the cerebral venous sinuses may have caused the continuously increased intracranial tension.

Ecker's case demonstrated *a new syndrome symptomatic of hydrocephalus of the cortex or of the subdural space following operative obliteration of the lateral sinus.*

The patient, a woman aged 21, complained, May 27, 1939 that her ears had been discharging for six years and that there had been exacerbation of pain for ten days, with tenderness over the left mastoid process.

The leukocyte count was 11,240, with polymorphonuclears 81 per cent.

Lumbar puncture showed colorless spinal fluid under a pressure of 20 cm. of water. Jugular compression caused an abrupt rise in the pressure. The following day a chill developed.

May 28 simple mastoidectomy performed on the left side revealed hemorrhagic mastoiditis. The wall of the lateral sinus was thickened and white. When the sinus was opened, there was free bleeding.

June 23 (twenty-five days after operation) there was a perforation of the right drum membrane, with slight discharge, and the patient complained of a continuous "fuzzy" sound in the right ear, of transient pain in the right eye and orbit, occasionally radiating over the right vertex, and pain in the left shoulder and arm.

There was choking of the disks (3 to 3.5 diopters), with hemorrhages and enlarged blindspots.

The spinal fluid was clear and under a pressure of more than 45 cm. of water

All attempts to reduce the increased intracranial pressure by restriction of the water intake and administration of magnesium sulfate failed. On July 12 ventricular estimation and ventriculography were attempted; 33 cc. of fluid was obtained from the left and 25 cc. from the right ventricle and replaced by air.

Roentgenograms revealed no air in the ventricles but a little air in the subarachnoid space under the burr holes (Davidoff-Dyke sign).

July 26 the left lateral ventricle was punctured, and then, with the patient sitting upright, a spinal puncture was done and approximately 150 cc. of fluid was removed and a small amount of air injected. During the removal of the fluid, air was heard being sucked through the needle into the left lateral ventricle. Roentgenograms showed the left lateral ventricle filled with air and normal in size, shape and position, but none of the rest of the ventricular system was revealed.

From July 29 through August 1 the spinal fluid pressure varied between 30 and 49 cm. of water.

Starting August 2, the patient was made to sit up in a chair continuously. The improvement in symptoms and the fall in the spinal fluid pressure were striking; the latter fell to 12.5 cm.

The patient was discharged September 7, disks flat.

Elimination of (1) Elevation of Systemic Venous Pressure and (2) Alteration of Osmotic Pressure as Factors.—Following L. S. Kubie's suggestion that *elevation of systemic venous pressure* might account for increased intracranial pressure, the venous pressure of the patient was determined on August 1. "In the antecubital fossa [in front of the elbow—which eliminated this factor] the venous pressure was 92 mm. (normal) and the circulation time was fourteen seconds."

Another possible systemic factor suggested was an *alteration in the osmotic pressure of the blood.* The serum protein was found "to be 7.4 Gm. per hundred cubic centimeters, a normal reading. There were 4.7 Gm. of albumin and 2.7 Gm. of globulin per hundred cubic centimeters of blood serum, and therefore there was no evidence of decreased osmotic pressure of the blood."

Comment (W. P. E.).—This case is somewhat similar to the one which I reported in 1906[149] and allows the same explanation except that Ecker's case was one of chronic advancing obliterating thrombosis which only reached the basal sinuses twenty-five days after operation while in my case venous stasis and extension thrombosis were caused immediately by the ligation of the jugular vein on the side of the large lateral sinus.

DIFFERENT CEREBRAL CONDITIONS IN CASES OF THROMBOSIS OF THE LATERAL SINUS

Inflammation of Dura the Cause of Papilledema in Cases of Sinus Thrombosis.

Friesner[150] concludes that the cranial venous sinuses are part of the dura. The one factor which is common to all these cases is inflammation of the dura. This is the possible cause of the papilledema seen frequently in otitic thrombosis of the lateral sinus.

Anatomic "Patterns" of Sinus Thrombosis That Cause Venous Back Pressure.

Woodhall[151] attempts to explain the different clinical pictures that are presented by venous back pressure the result of thrombosis of the

149. Eagleton, W. P.: Circulatory Disturbances Following Ligation of the Internal Jugular Vein in Sinus Thrombosis, with Report of Case, Arch. Otolaryng. **25**:91-102 (April) 1906; footnote 21, case 23, p. 129; abstracted, Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in the Understanding of Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1940, p. 24.

150. Friesner, I.: Analysis of Ocular Fundus Changes in Otitic Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **50**:32-37 (March) 1941.

151. Woodhall, B.: Anatomy of the Cranial Blood Sinuses with Particular Reference to the Lateral, Laryngoscope **49**:966-1010 (Oct.) 1939.

lateral sinus and the corresponding anatomy of the torcular Herophili and its contiguous sinuses. Patients with venous thrombophlebitis may be divided into five groups:

1. Common pool type.
2. Plexiform type, with adequate intercommunication between the lateral sinuses.
3. Ipsilateral type, in which the longitudinal sinus runs to one side and the straight sinus to the opposite side.
4. Unilateral type, in which the longitudinal and the straight sinus empty into one lateral sinus.
5. Occipital type, which is the "persistence of a large single or paired occipital sinus with large marginal sinuses, comparable to the pattern . . . . common in the fetus or during the first few years of life."

(He studied 100 preparations.)

Supposed Serous Meningitis Following Extensive Thrombphlebitis of Large Venous Sinuses from Apical Necrosis Presenting No Localized Symptoms Until a Terminal Meningitis.—Woodhall reports this case: Three months before admission the patient, a man 46 years old, had an infection of the upper respiratory tract and a sore throat, followed by acute otitis media on the right side, associated with headache and swelling of the right side of the neck. After myringotomy the ear drained for ten days. For a month there was pain over the right side of the face, involving the eye and the upper and lower teeth, and there was a peripheral palsy of the seventh nerve on the right side, which disappeared completely in ten days. One week before admission he had a third sore throat. The next day a tender mass developed beneath the angle of the right jaw.

Examination showed the right ear drum obscured by edema in the region of Shrapnell's membrane. Hearing was grossly normal. There was a tender cervical lymph node at the angle of the right mandible.

Both fundi showed papilledema, the right with loss of the physiologic cup, overfilling of the veins and exudation about the disks.

Roentgenographic plates suggested infection of the right mastoid process extending into the petrous pyramid.

A ventricular air injection was done. The initial pressure was 400 mm. of water, with the patient in the recumbent position; on compression of each jugular vein there was a rise of 100 mm. of water. The ventricular fluid showed 3 cells; the protein content was 25 mg. per hundred cubic centimeters. The ventriculograms were normal.

The clinical impression was "serous meningitis."

Right subtemporal decompression was performed. The brain was found to be edematous with excessive fluid.

Simple mastoidectomy was performed on the right side; the cells were filled with granulation tissue.

Postoperatively there was definite regression of the papilledema.

Next day the patient had fulminating meningitis and died within twenty-four hours. Pneumococcus type III was isolated from the spinal fluid.

The right temporal bone was examined post-mortem. The base of the petrous pyramid had a purplish red coloration; on the inferior, posterior and mesial parts there was a horizontal perforation 1 cm. in length. A probe passed into this perforation communicated with the mastoidectomy wound. The superior petrosal sinus, the transverse sinus on the right side, the torcular Herophili and the superior longitudinal sinus all contained thrombi. In the superior longitudinal sinus the thrombi varied from place to place. The author calls it an "ipsilateral sinus pattern."

Slowly Advancing Thrombophlebitis of the Cerebral Sinuses and Cortical Vessels with Symptoms Simulating Multiple Tumor of the Brain.

Otogenous phlebitis of a lateral sinus may have as its only symptoms headache and slight fever, and even these may be absent if the sinus is only partially occluded.

In a case of "otitic thrombosis of the cerebral sinuses and veins" reported by Keschner and Davison [152] the symptoms during life were thought to be due to multiple tumor of the brain. Autopsy showed, however, that the symptoms resulted from thrombosis and recent hemorrhages in the brain, especially in the regions drained by the right and the left middle and posterior cerebral veins.

The patient, a man 46 years old, had had an intermittent purulent discharge from the right ear for three years. April 23, 1937 he fell, striking his back and the back of his head. There were no immediate symptoms referable to the head.

Two weeks later, urination became frequent and painful, and June 2 he had hematuria. June 9 left ureteronephrectomy occasioned a pathologic diagnosis of "tuberculous pyelonephritis and ureteritis."

In October he resumed his occupation for four months. On April 25, 1939 (one year after the fall and nine months after the urologic operation) he suddenly had weakness of the entire left upper extremity with numbness and tingling in the hand and fingers.

May 4 he complained of generalized headache. Examination showed: perforation of the right middle ear, with slight discharge, and deafness; weakness of the left side of the face "of supranuclear type"; left hemiparesis of the upper motor neuron type, more marked in the upper extremity; hypesthesia and hypothermesthesia of the left upper extremity; a Babinski sign on the left side; Hoffmann's sign bilaterally.

On the fifth day after an encephalographic examination he was nauseated and vomited. On the sixth day there were urinary incontinence, nuchal rigidity, a bilateral Kernig sign, ankle clonus and a Babinski sign. His temperature was 100.4 F. On lumbar puncture the spinal fluid was hazy and under a pressure of 400 mm. of water; it contained 12 lymphocytes per cubic millimeter and 67 mg. of sugar, 73.4 mg. of protein and 794 mg. of chlorides per hundred cubic centimeters. Smear and cultures revealed no bacteria. The Ayala index was 4.5.

Ten minutes after the spinal puncture a generalized convulsion occurred and another convulsion two hours later.

---

152. Keschner, M., and Davison, C.: Otitic Thrombosis of the Cerebral Sinuses and Veins Simulating Multiple Brain Tumors, Arch. Neurol. & Psychiat. **47**:428-437 (March) 1942.

On the ninth day after the encephalogram had been made, he had repeated convulsions; his temperature was 103 F. and the respiratory rate 64. There were increased signs of meningeal involvement. Lumbar puncture yielded hazy fluid under a pressure of 460 mm. of water; it contained 29 lymphocytes per cubic millimeter; the Pandy reaction was positive; the tryptophan test was positive; there was definite pellicle formation.

Diagnostically the authors observed that "the objective neural signs were those of a lesion in each cerebral hemisphere, and, although there were no evidences of meningitis or of intracranial hypertension, the history of tuberculosis suggested the possibility of multiple tuberculoma of the brain. Intracranial injection of air disclosed moderate internal hydrocephalus, with no displacement of the ventricular system, and more or less symmetric bilateral cortical atrophy."

Death occurred on the ninth day after encephalography and development of symptoms of meningeal involvement.

Necropsy.—The dural sinuses were filled with clots, slightly adherent. The superior longitudinal and right sigmoid sinuses contained a thrombus with slight organization. Microscopic sections of the superior longitudinal sinus showed a mural thrombus with beginning organization.

The cerebral convolutions were partially obliterated. The subarachnoid cortical vessels were distended, most of them being thrombosed. In the left parieto-occipital region was a magenta-colored softening. Small hemorrhagic areas also involved part of the motor, superior parietal and temporal convolutions on the right side.

Microscopic examination of tissue from the right ear showed necrotic bone, in which the organ of Corti was identified. Fragments were filled with lymphocytes, plasma cells and endothelial cells. There were also a few polymorphonuclear leukocytes and congested and proliferated blood vessels.

The histologic examination indicated that the venous thrombi and hemorrhages were secondary to an infectious and thrombotic process in the dural sinuses, especially in the right lateral and the superior longitudinal sinus.

Probable Sequence of Advanced Pathologic Processes, Which Had Existed for Several Months.—The sequence was probably as follows: Chronic otitis media on the right side, with osteomyelitis of the petrous portion of the temporal bone, caused (*a*) thrombosis of the right lateral sinus with (*b*) retrograde extension of the thrombus to involve the superior longitudinal sinus; the involvement of this sinus (*c*) by obstructing the tributary cerebral veins interfered with the venous, and ultimately with the arterial, circulation of the brain, which led in turn (*d*) to hemorrhages and (*e*) softening of areas in the left parieto-occipital region and small hemorrhages in the motor, superior parietal and temporal convolutions of the right hemisphere. (*f*) The terminal meningeal reaction and subarachnoid hemorrhages were secondary to thrombosis and rupture of the cerebral blood vessels.

SENSITIVITY OF DURA AND LOCATION OF PAIN IN THE DIAGNOSIS OF NONSUPPURATIVE INTRACRANIAL LESIONS

Distribution of Sensory Nerves in the Cranium.

Greater Superficial Petrosal Nerve and Anterior and Posterior Ethmoid Nerves.—From observations in monkeys Chorobski and Penfield in 1932 suggested that the greater superficial petrosal nerve may

supply sensory fibers to the dura. Penfield and McNaughton [153] point out that some fine nerves come from the anterior and posterior ethmoid nerves (to the falx cerebri and the superior sagittal sinus).

Glossopharyngeal Nerve.—They state: "It is difficult to trace . . . dural nerves . . . to their origin in the vagus trunk within the jugular foramen, where the vagus is closely associated with the glossopharyngeal and accessory nerves; it may be that some twigs are derived from the glossopharyngeal nerve, as stated by Stöhr.[154]

"A few fine nerves are sometimes seen branching in the dura about the dural openings for the hypoglossal nerve. According to some anatomists these are hypoglossal fibers. Poirier, Charpy and Nicolas [155] described them as fibers from the lingual nerve."

Nerves Entering Petrous Bone.—The nerves entering the petrous bone are presumably sensory fibers destined for the mastoid cells and the middle ear.

Nerves Following Blood Vessels.—Penfield and McNaughton observed that the nerves tend to be distributed about blood vessels and along the large venous sinuses, branching off into the dura itself.

The main dural supply seems to be made up of nerves containing vasomotor (sympathetic) fibers for the meningeal arteries, together with many sensory (trigeminal, vagus and other) fibers which probably end in, or close to, the walls of meningeal vessels and sinuses.

The arrangement of the dural nerves, particularly those in the middle fossa, is not necessarily the same on the two sides. There also seem to be differences in the number of dural nerves from person to person.

Summary.—1. The trigeminal nerve supplies the cerebral dura everywhere except in the posterior cranial fossa. 2. The middle meningeal arteries carry with them fibers which emerge from the ganglion with the second and third divisions and less constantly with the first division. 3. The superior sagittal (or longitudinal) sinus is supplied mainly from the first division of the trigeminal nerve (through the nervus tentorii and a branch of the anterior ethmoid nerve from each side). 4. The transverse sinus (superior wall) is supplied by the tentorial nerve. 5. The inferior wall of the transverse sinus and the dura of the posterior fossa are supplied by the tenth cranial nerve, smaller branches arising possibly from the ninth and twelfth nerves.

153. Penfield, W., and McNaughton, F.: Dural Headache and Innervation of the Dura Mater, Arch. Neurol. & Psychiat. **44**:43-74 (July) 1940.

154. Stöhr, P., Jr.: Mikroscopische Anatomie des vegetativen Nervensystems, Berlin, Julius Springer, 1928, p. 177.

155. Poirier, P.; Charpy, A., and Nicolas, A.: Traité d'anatomie humaine, Paris, Masson & Cie, 1921, vol. 3, pp. 316-318.

Location of Pain in Intracranial Structures.

Foerster [156] in 1927 reported that the dura is sensitive particularly at the base of the skull and close to larger branches of the middle meningeal artery, while the dura of the convexity is less sensitive. The tentorium is very sensitive, but not the pia mater or the brain substance.

In 1934 Penfield [157] reported on the sensitivity of the dura and other intracranial structures and on location of pain in them:

"*Cerebral Veins and Arteries.*—Electrical stimulation of the middle cerebral artery near the circle of Willis may produce pain in the ipsilateral temporal region or above the ipsilateral eye. . . ."

"*The Dura.*— . . . Pressure . . . of the middle meningeal artery or its branches causes sharp pain . . . localized to the area of stimulation or . . . referred to the ipsilateral temporal region. . . .

"*Walls of the Dural Sinuses.*—The walls of the sinuses, and sometimes the large veins entering the sinuses, are found to be extremely sensitive. Pain produced from the dural sinuses, like that arising from pressure on the falx and tentorium, is apt to be referred to a distance. . . .

"*Character of Pain.*—The sinuses are sensitive to stimulation by pressure, traction, heat and electricity. All these forms of stimulation produce . . . pain which is described by the patient as sharp or pressing, or as 'headache' . . ."

Headache Due to Irritation of the Dura.—"It has . . . been suggested that headaches are due to irritation of the dura and its septums, particularly the area about branches of the meningeal arteries; . . . Other sources of headache . . . suggested are the cerebral arteries [158] and the walls of the third and lateral ventricles of the brain"[159] . . . (Penfield and McNaughton).

Penfield and McNaughton found "no fibers passing from the trigeminal nerve to the cerebral arteries." They suggest that if the largest cerebral arteries are truly sensitive to pain, the greater superficial petrosal nerve may carry the sensory fibers. "The efferent fibers of this nerve are parasympathetic and serve as vasodilators for the cerebral vessels, but there are some afferent fibers the ganglion cells of which are situated in the geniculate ganglion. . . . "

The nerve supply of the dura and sinuses of the posterior fossa arise "chiefly in the vagus nerve, with addition of fibers . . . from the hypoglossal, and possibly from the glossopharyngeal, nerve."

---

156. Foerster, O.: Die Leitungsbahnen des Schmerzgefühls und die chirurgische Behandlung der Schmerzzustande, Berlin, Urban & Schwarzenberg, 1927, pp. 10-11.

157. Penfield, W.: A Contribution to the Mechanism of Intracranial Pain, A. Research Nerv. & Ment. Dis., Proc. (1934) **15**:399-416, 1935.

158. Fay, T.: The Mechanism of Headache, Tr. Am. Neurol. A. **62**:74-77, 1936.

159. Elsberg, C. A.: The Intracranial Pressure in Health and Disease, A. Research Nerv. & Ment. Dis., Proc. **8**:3-6, 1929.

Referred Pain.—"The ache which results from stimulation of the dural sinuses is usually referred to a distance from the point of stimulation. [The direction of this referred pain is anatomic.] The right lateral wall of the superior sagittal (longitudinal) and the straight sinus and the upper wall of the right transverse sinus are innervated by the ophthalmic division of the right trigeminal nerve, and these walls are easily stretched either by pressure or by traction exerted directly on the sinus."

Penfield and McNaughton reported that in their case 6 of dural headache and "atypical trigeminal neuralgia" migraine was relieved by rhizotomy (rhiza-root), viz., section of the posterior spinal root for the relief of pain or spasticity (Dana's operation).

The patient, a woman aged 38, had a family history of occurrence of migraine. Her attacks of pain began at 8 years of age and came to have a periodicity of about twelve days. She was free from them only when pregnant. In 1929 she suffered from headaches in the right frontal region, radiating to the right shoulder. The attacks were preceded by a sense of fatigue, facial pallor and a sensation of fulness in the nose.

Injections into the sphenopalatine ganglion gave only partial relief.

Cervical and upper thoracic sympathetic ganglionectomy and perivascular sympathectomy of the carotid and vertebral arteries on the right side took away the spread of the head pain to the neck and the shoulder but produced for a period of about six months neuritic pain in the right arm and pain in the parotid region on eating. The headache continued.

In December 1929, procaine hydrochloride was injected into the gasserian ganglion during an attack. The needle was inserted into the third division at the foramen ovale. The lower lip and the tongue became numb first, but there was no effect on the headache. Next, the upper lip and the nose became numb, with no alteration in the headache. As the area of distribution of the ophthalmic division became numb the patient remarked suddenly: I have nothing to feel the pain with," and her headache vanished. It returned when the analgesia had worn off.

Consequently, subtotal posterior trigeminal rhizotomy was carried out so as to sever the roots of the ophthalmic division. There resulted complete analgesia in the distribution of the first division and partial loss of pain in that of the second division.

In the ten years that have elapsed since this operation there has been no recurrence of headache.

THE SYMPATHETIC SYSTEM AND THE EAR

Horner's Syndrome Due to Chronic Otitis; Intraconjunctival Instillation of Epinephrine Hydrochloride in Diagnosis.[160]

Horner's symptom complex (Johann Friedrich Horner, Zurich, Switzerland, ophthalmologist, 1831-1886) consists of ptosis, miosis, anhidrosis and enophthalmos due to paralysis of the cervical sym-

160. See Stedman, T. L., and Garber, S. T.: Stedman's Practical Medical Dictionary, ed. 14, Baltimore, William Wood & Company, 1939, plate XX, p. 1084.

pathetic system. Hubert [161] indicates that there have been but few observations on involvements of the sympathetic nervous system resulting in Horner's syndrome in the course of chronic lesions of the tympanic cavity. He reports a case and states that he knows of no similar case recorded in the American literature.

There is experimental evidence that in animals destruction of the tympanic cavity will produce Horner's syndrome.

Anatomic Explanation of Horner's Syndrome from Disease of the Ear.—Piquet and Coulouma's [162] investigations of the distribution of the nerve fibers at the level of the promontory of the middle ear and of the relationship of the sympathetic plexus in the vertical portion of the carotid canal in the petrous portion of the temporal bone show that: 1. Often several filaments from the carotid sympathetic plexus detach themselves and penetrate the cavity of the middle ear. They travel to the anterior part of the promontory underneath the mucous membrane and sometimes in the depth of the bone of the anterior wall to the petrosa. This explains why Jarcho and Root [163] observed sympathetic ocular phenomena following labyrinthectomy in cats. 2. The sympathetic filaments coming from the carotid plexus and traversing the tympanic cavity rejoin this plexus and finally reach the dilator muscle of the pupil by accompanying branches of the long ciliary nerves. The majority and sometimes all the sympathetic ocular fibers continue along the carotid plexus without entering the cavity of the middle ear.

Hubert's Case.—G. S., aged 37 years, complained of pain in the right half of the head and discharge from the right ear. Eight years previously radical mastoidectomy was performed. After the operation she had (*a*) drooping of the right upper lid, (*b*) constriction of the right pupil, (*c*) narrowing of the palpebral fissure and (*d*) apparent enophthalmos of the right eye—Horner's syndrome complex, due to paralysis of the cervical sympathetic system. There was partial atresia of the right auditory canal. A roentgenogram of the right mastoid process suggested cholesteatoma.

Diagnostic Use of Epinephrine.—Loewi [164] in 1908 showed that normally when a solution of epinephrine hydrochloride, 1 to 1,000, is instilled into the conjunctival sacs, no mydriasis will ensue. However, if the sympathetic nervous system is rendered hyperirritable, as it may be in persons with exophthalmic goiter or diabetes, dilatation of the pupils will become apparent.

The epinephrine test is useful in the early diagnosis of malignant tumors of the nasopharynx. In the diagnosis of Horner's syndrome the epinephrine test may be the only deciding factor. A dilatation of the

161. Hubert, L.: Horner's Syndrome with Chronic Purulent Otitis Media: Demonstration of a Diagnostic Test, Laryngoscope **53**:46-49, (Jan.) 1943.

162. Piquet, J., and Coulouma, P.: Les signes oculo-sympathiques dans les otites et leurs raisons anatomiques, Ann. d'oto-laryng., September 1935, pp. 1003-1013.

163. Jarcho, D. W., and Root, W. S.: The Relation of Labyrinthectomy to Sensitization of the Nictitating Membrane of Cat, Am. J. Physiol. **128**:526-531 (Feb.) 1940.

164. Loewi, O.: Ueber eine neue Funktion des Pankreas und ihre Beziehung zum Diabetes mellitus, Arch. f. exper. Path. u. Pharmakol. **59**:83-94, 1908.

pupil after instillation of epinephrine hydrochloride indicates that the postganglionic fibers are involved.

Technic of Hubert's Diagnostic Test with Cocaine and Epinephrine.—A 4 per cent solution of cocaine hydrochloride instilled into the conjunctival sac of a normal person will cause dilatation of the pupil. In the presence of Horner's syndrome the dilatation will not occur. By instilling a solution of epinephrine hydrochloride into the affected eye one can tell with reasonable certainty whether the lesion is (1) in the superior cervical ganglion and its postganglionic fibers or (2) in the preganglionic fibers and the central nervous system. In the first instance the pupil will dilate, the drooping of the eyelid will disappear and the eyeball will protrude slightly.

Hubert explains that:

". . . denervated smooth muscles become sensitive to the circulating epinephrine in the blood and . . . will become still more sensitive when this substance is directly applied by conjunctival instillation. If the dilator muscle of the pupils and the other bundles of smooth muscles of the eye are cut off from their nerve connection with the superior cervical ganglion (*i. e.*, if the lesion is in the ganglion or its postganglionic fibers), epinephrine hydrochloride instilled into the conjunctival sac will act directly on the denervated muscle fibres and cause not only dilatation of the pupil but elevation of the lid and an apparent slight protrusion of the eyeball." [He states that this reaction was obtained in the case which he reported] "showing that the lesion was in the postganglionic fibres.

". . . The lesion causing this condition [Horner's syndrome] may be in the superior cervical ganglion, the preganglionic and postganglionic fibres or the central nervous system."

TUMORS OF THE EIGHTH (ACOUSTIC) NERVE

Neurofibroma of the Seventh (Facial) Nerve Involving the Mastoid Process.

Neurofibromas occur most commonly in small cutaneous nerves; however, a neurofibroma may arise from any nerve trunk.

In the case reported by Pastore and Williams [165] the neurofibromatous mass with lipoidal degeneration apparently originated in the facial canal four years previously and later involved the mastoid process.

The patient, aged 59 years, had bitten the left side of his upper lip accidentally four years previously (1933); this was followed by drooping of the lip. Within a month complete paralysis of the left facial nerve developed. In June 1937 he noted soreness and pain in the left mastoid process with increased deafness and pounding in the left ear. A polyp in the left auditory canal was removed. Shortly afterward tissue was again removed from the left auditory canal, but soon recurred. In November the patient had complete paralysis of the left facial nerve; the muscles of the left side of the face showed a complete reaction of degeneration.

165. Pastore, P. N., and Williams, H. L.: Neurofibroma of the Seventh Nerve Involving the Mastoid, Proc. Staff Meet., Mayo Clin. **14**:524-528 (Aug. 16) 1939.

and the muscles of the left side of the forehead were atrophic. A polypoid granulomatous mass could be seen protruding into the left concha. There was complete nerve deafness on the left side.

Roentgenograms revealed irregular destruction of the left mastoid process over the lateral sinus.

Biopsy showed that the tissue filling the external auditory canal consisted of an inflammatory ulcer and granulation tissue.

Radical mastoidectomy was performed on the left side. The mastoid cells contained a soft yellowish material, which had eroded the bone and broken through at the mastoid tip and anteriorly just above the posterior wall of the external auditory canal; it had also extended deep into the pyramid and into the middle ear. Most of the labyrinth had been absorbed, as there was a cylindric channel following the course of the facial nerve anterior to the cochlea.

On the third and fourth days after operation 12,000 milligram hours of radium was administered in divided doses.

Convalescence was uneventful.

Altmann [166] in 1935 and 1937, Tremble and Penfield [167] in 1936 and Gnassi and Borrone [168] in 1938 described tumors affecting the seventh nerve and their treatment.

Radioresistance of Neurofibroma of the Facial Nerve.—Desjardins [169] stated that since neoplasms which arise from tissues known to be resistant to roentgen rays are usually resistant to radium, and since nerve and fibrous tissues are very resistant to roentgen rays, it is reasonable to assume that neurofibroma is also resistant to these rays and to radium.

Hereditary Deafness with Tumor of the Acoustic Nerve.

In 1930 Fraser and Gardiner [170] reported a family in which von Recklinghausen's disease in the form of bilateral tumors of the acoustic nerves had been transmitted as a dominant mendelian trait through five generations. At the time of the original report 38 of the 217 members of the family were affected. Necropsy performed on 2

166. Altmann, F.: Ueber neurogene Tumoren des absteigenden Facialisteiles, Monatschr. f. Ohrenh. **69**:1032-1048 (Sept.) 1935; Zur Kenntnis der Tumoren des absteigenden Fazialisteiles, ibid. **71**:1287-1292 (Nov.) 1937.

167. Tremble, G. E., and Penfield, W.: Operative Exposure of the Facial Canal with Removal of a Tumor of the Greater Superficial Petrosal Nerve, Arch. Otolaryng. **23**:573-579 (May) 1936.

168. Gnassi, A. M., and Borrone, M.: Schwannoma, Arch. Otolaryng. **27**: 766-772 (June) 1938.

169. Desjardins, A. U.: Radiotherapy (Roentgen Rays; Radium), J. A. M. A. **105**:2064-2071 (Dec. 21); 2153-2161 (Dec. 28) 1935.

170. Fraser, J. S., and Gardiner, W. T.: Tumors of the Eighth Nerve, J. Laryng. & Otol. **45**:361-366 (May) 1930; abstracted, Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **26**:690-735 (Dec.) 1937; Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1941, reprint (1937), p. 90.

members revealed the bilateral acoustic neurofibromas. After publication of the first report, symptoms of a tumor of the spinal cord developed in one of the affected members. This tumor proved to be a neurofibroma.

The present report is the third concerning this family.

Case 1[171] (total deafness for nineteen years and blindness for twelve years, with necropsy revealing bilateral acoustic neurofibromas and a small meningioma adherent to one of the growths).—A woman, aged 40 when she died in 1935, had been completely deaf since the age of 21; the onset of deafness had been gradual and associated with tinnitus; otherwise, she was well until the age of 28. After the birth of a child she had headaches, vomiting and rapid failure of vision.

In 1923 she was totally deaf and blind. She had choking of the optic disks and paralysis of the right facial nerve of the peripheral type. Vestibular tests elicited no response except possibly from the horizontal canal. Gait was ataxic. The spinal fluid pressure was 32 mm. of mercury.

In 1929 she could walk only with support because of severe ataxia of the arms and legs. There was marked spontaneous nystagmus. Both corneas were anesthetic. Optic atrophy was complete. Death occurred on July 25, 1935.

Autopsy revealed tumors occupying the cerebellopontile angles.

Description of the Tumors.—The external surface of the growth on the right side measured 6.5 by 7.5 cm.; the growth was nodular and light grayish yellow. On its medial aspect it was in contact with the brain stem and the cerebellum. It was well encapsulated and easily shelled out. The seventh and eighth nerves entered the tumor substance. The tumor was enveloped by a thin transparent membrane, which appeared to be the pia-arachnoid. The growth on the left was about half the size of that on the right. Section revealed no area of reddish gray tissue; such an area was found in the growth on the right.

Histologically, the bulk of the tumor on the right was a fairly typical neurofibroma, complicated by the presence of a second growth, a meningeal tumor,[172] which had been represented in the gross specimen by the small reddish gray area.

Sections through the meningeal tumor disclosed that this growth had arisen in the leptomeninges which enveloped the larger tumor. Microscopically, this tumor corresponded with that classified by Cushing and Eisenhardt as type II, variant 3.[173]

Diagnosis: bilateral acoustic neurofibroma with an associated meningioma on the right side.

Case 3 (deafness of thirty-two years' duration, with necropsy revealing small bilateral acoustic neurofibromas).—Although acoustic nerve tumors of the small size of these have been described, in no instance have they been bilateral. The history suggests both tumors had been present for more than thirty years. Bilateral acoustic neuromas tend to a more slowly progressive course than do the solitary ones.

---

171. Gardner, W. J., and Turner, O.: Bilateral Acoustic Neurofibromas: Further Clinical and Pathologic Data on Hereditary Deafness and Recklinghausen's Disease, Arch. Neurol. & Psychiat. **44**:76-99 (July) 1940.

172. Gardner, W. J., and Turner, O. A.: Multiple Intracranial Tumors: A Discussion of the Relationship of Meningeal to Acoustic Tumors and a Report of a Case, J. A. M. A. **113**:111-113 (July 8) 1939.

173. Cushing, H., and Eisenhardt, L.: Meningiomas: Their Classification, Regional Behavior, Life History, and Surgical End-Results, Springfield. Ill., Charles C Thomas, Publisher, 1938.

The patient, a woman, was 58 years old at the time of death in September 1930; progressive deafness began at the age of 26, the right ear being affected first. Tinnitus began at 44, when she sustained a fracture of the nose. Occasional dizziness and staggering began at 49. At 56 she was totally deaf, and there was no response from either vestibular nerve in the turning or the douching test.

The patient's son had likewise been deaf for thirty years.

Description of Tumors.—Autopsy revealed in each cerebellopontile angle a small tumor, which had arisen from the eighth nerve. The tumor on the right measured about 1.5 cm. and was flattened against the porus acusticus, to which it was adherent. The tumor on the left measured about 1 cm. It filled the porus acusticus.

Microscopically both tumors were typical neurofibroma, with a definite but thin capsule composed of collagenous tissue. Numerous moderate-sized blood vessels were scattered throughout the tissue.

Tumor of the Acoustic Nerve Situated Within the Petrous Bone.

In the case reported by Adelstein and Anderson [174] the tumor disclosed at operation was completely covered by the greatly thinned bony wall and the overlying dura. The case is unique in that the tumor arose within the internal auditory canal and expanded by erosion of the bony canal.

The patient was a girl aged 15 who six years prior to hospitalization had gradual onset of tinnitus and deafness of the right ear. The tinnitus never increased, but the impairment of hearing increased to a loss of 50 per cent.

Three years before admission to the hospital weakness of the right side of the face developed, and within a year the right side of the face was entirely paralyzed, but she could partially close the right eyelid by strongly closing the left eye.

Audiometric examination demonstrated perception deafness of the right ear, with loss of 50 per cent in all ranges. Stimulation of the right ear with cold water elicited no response from the horizontal semicircular canal and a delayed reaction from the vertical canals.

Cerebellar signs were minimal; there were slight dysdiadokokinesis and asynergy in the right upper extremity. The deep reflexes were somewhat greater in the right extremities than in the left. Spinal puncture revealed a pressure of 260 mm. of water, with normal dynamics. The serologic tests were negative. Roentgenograms of the skull showed erosion of the ridge of the right petrous bone in the region of the internal acoustic meatus.

The peroperative diagnosis was: an acoustic neuroma or a cholesteatoma in the right cerebellopontile angle.

Operation.—The bony floor of the posterior fossa was removed superiorly to the torcular Herophili and inferiorly to include the posterior half of the ring of the formen magnum. The posterior horns of both lateral ventricles were tapped. The dura over the cerebellum was opened, and the straight sinus was tied with silk. The cisterna magna was emptied. In the right petrous bone a large, hard eminence was discovered. The bony shell was opened, and as large an amount of tumor tissue as possible was removed.

174. Adelstein, L. J., and Anderson, F. M.: Tumor of the Acoustic Nerve Within the Petrous Bone: Operative Removal, Arch. Neurol. & Psychiat. **51**: 268-270 (March) 1944.

Three months later a spinal accessory–facial nerve anastomosis was made on the right side.

Description of Tumor.—Microscopic study disclosed a rather dense fibrous stroma with some tendency to bandlike arrangement and pseudopalisading of cells. In several areas the connective tissues had undergone a mild degree of hyalinization. Diagnosis: neurofibroma.

Comment (W. P. E.).—The vestibular reactions were suggestive of increased intracranial pressure.[57]

Ventriculography to Determine the Degree of Hydrocephalus.

Schönbauer [175] discusses the importance of making a preoperative ventriculogram when the presence of a tumor of the acoustic nerve is suspected.

With the ventriculogram one can determine the degree of hydrocephalus and also the possibility of an occlusion in the region of the aqueduct.

High Postoperative Mortality in Cases of Attempted Total Removal of Tumor of the Acoustic Nerve.

Schönbauer exposes the tumor and attempts to remove it in toto. In a series of 56 cases he had 17.9 per cent immediate postoperative fatalities (6 patients died of pneumonia, 7 of respiratory paralysis and 1 of meningitis). Twenty-five of the remaining 42 patients were followed up; 9 were alive longer than three years, 9 were alive less than three years, and 7 had died within the three year interval.

Operative Treatment of Tumor of the Acoustic Nerve.

Extirpation of Tumor of the Acoustic Nerve Within the Internal Auditory Meatus by Dandy's Method with Modifications.—Horrax and Poppen [176] removed tumors by complete extirpation in 35 patients. The mortality for total extirpation of primary and recurrent tumors combined was 17.8 per cent.

Horrax states that the tumors always extend into the internal auditory meatus to a considerable depth. Therefore, in order to remove every cell of the tumor, he always curets the meatus. It has usually also been found necessary either to chip off the bony ridge over the meatus or to make burr openings with a small drill lest one break into the mastoid cells. Not only is the meatus curetted; it is coagulated.

Translabyrinthine Operation for Tumor of the Acoustic Nerve.—Güttich [177] describes the two methods of approach to acoustic tumors:

175. Schönbauer, L.: Chirurgische Behandlung der Acusticusgeschwülste, Ztschr. f. Hals-, Nasen- u. Orenh. **46**:118, 1939; abstracted, Arch. Otolaryng. **33**:1081 (June) 1941.

176. Horrax, G., and Poppen, J. L.: Experience with the Total and Intracapsular Extirpation of Acoustic Neuromata, Ann. Surg. **110**:513-523 (Oct.) 1939.

177. Güttich: Surgical Treatment of Acoustic Tumors, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:122, 1939; abstracted, Arch. Otolaryng. **33**:1082 (June) 1941.

(1) the translabyrinthine (Panse) through the domain of the auditory nerve, and (2) the paralabyrinthine (von Seiffert). He has performed eleven translabyrinthine and two paralabyrinthine operations, with three immediate postoperative fatalities.

Technic.—In Panse's translabyrinthine operation the labyrinth is completely resected, and the facial nerve is exposed. The nerve is followed (Panse) to the geniculate ganglion, then to the posterior fossa and to the internal auditory meatus. After resection of the labyrinth has been completed, the posterior surface of the petrous pyramid is exposed. To reach the cerebellopontile angle he incises the dura from the sigmoid sinus to the internal auditory meatus.

(*To Be Continued*)

Abstracts from Current Literature

Ear

HEARING CONSERVATION AT THE COLLEGE LEVEL. RAIDA DEDOMENICO, J. Speech Disorders **10**:275, 1945.

At the University of Iowa, in the fall of 1944, 855 entering students were tested with respect to hearing acuity by means of the sweep check technic, the Maico Audiometer Model D-5 being used; 787 were found to have normal hearing, while 68 were suspected of having hearing losses, with 14 of these having severe colds at the time of testing. Of the 68, 12 failed to report back. Of those reporting back, 34 were found to have hearing losses with the threshold acuity method, and 21 of the 34 completed the otologic examination. Positive medical findings were found in all of these 21. A group of 26 hard of hearing freshmen, included in the total group of 34, was matched with a control group of 52 normal hearing freshmen as to race, sex, age and freshman examination scores, and the two groups were compared as to speech performance and scholastic record for the fall semester of 1944. Significant differences were found between these groups in regard to the ratings for articulation in speaking and oral reading and less marked differences with respect to voice. Hard of hearing students presented misarticulations to a significantly greater degree than normal hearing students. There were no other significant differences between the two groups.

PALMER, Wichita, Kan.

MYRINGITIS GRANULOSA. JOHN J. HOCHFILZER, Laryngoscope **55**:509 (Sept.) 1945.

Myringitis granulosa is a supposedly rare disease, although Hochfilzer observed it a number of times during physical examinations among Army inductees.

Sense of fulness, itching and occasional discharging of the ear are the symptoms. There is no loss of hearing. The ear contains debris and foul-smelling discharge. Much of the tympanic membrane may be nearly normal in appearance, while some of it is covered with raspberry-like granulations. There is no perforation. The main condition from which to differentiate it is chronic otitis media.

The pathologic and etiologic aspects of myringitis granulosa are discussed. Fungi undoubtedly are at least partly responsible. The treatment is outlined. A report of 7 cases is included.

HITSCHLER, Philadelphia.

THROMBOPHLEBITIS OF THE LATERAL SINUS, COMPLICATED BY PREGNANCY, WITH COMPLETE RECOVERY. W. E. SAUER and DORIS SURLES WOOLSEY, Laryngoscope **55**:519 (Sept.) 1945.

The case of thrombophlebitis of the lateral sinus reported was complicated by pregnancy, toxic hepatitis and pyelitis. Treatment with penicillin as well as surgical intervention brought about a cure. Three months later the patient delivered a normal healthy baby. Details of the case are given.

HITSCHLER, Philadelphia.

CARE OF THE INJURED EAR. G. V. WEBSTER, Surgery **18**:515 (Oct.) 1945.

In cases of injury of the external ear, Webster stresses the desirability of saving all the tissue possible and, if there is no considerable loss of tissue, immediate repair. In the latter case the field should be kept moist with a simple dressing until operative reconstruction is possible. For the operation he uses a flap of retroauricular skin, moved forward, or a tubed pedicle flap of cervical skin

and subcutaneous tissue, and sometimes a combination of these. When needed, according to the judgment of the operator, cartilage supports or metallic wires or strips are added at suitable stages of the procedure. The results have been usually satisfactory.

SELTZER, Philadelphia.

Nose

RHINOLOGY IN CHILDREN: RÉSUMÉ OF AND COMMENTS ON THE LITERATURE FOR 1944. D. E. S. WISHART, Laryngoscope **55**:381 (Aug.) 1945.

The common cold is caused by a virus. Secondary invaders produce the complications. The peaks of incidence of colds are in September-October, January-February and April-May. The incubation period is twenty-four to thirty-six hours. The spread of colds could be controlled by isolation of the infected persons. Other methods of prevention have been disappointing. Vaccines and vitamins have not been of value. Intranasal spraying of vaccine, however, has at least a scientific basis. Sulfonamide compounds are effective as preventives, and codeine and papaverine seems to shorten the duration of the cold. Crowe believes that irradiation to eliminate excessive lymphoid tissue is of great value; he believes that this reduces the number of colds and infections of the upper respiratory tract.

Many methods of treatment are mentioned. Frequent local use of the sulfonamide drugs may be of value.

Use of nonirritating shrinking solutions, spot suction, irrigation with Ringer's solution and not allowing the bedroom temperature to be below 50 F. are advocated by Schall as methods of treatment.

Cold vaccines, whether given by mouth or by hypodermic injection, have not proved effective.

Treatment of sinusitis in children should be directed to the region of the middle turbinate, for it is in this region that many of the sinuses drain. Wishart condemns interference with the inferior turbinate, washing the maxillary sinus by the natural ostium and some other rather widely used procedures. He stresses *personal* after-care following sinal operations.

Tonsils should be removed only when so enlarged that they interfere with swallowing or when they are chronically infected. Adenoids should be removed when they obstruct the air passages, the sinuses or the eustachian tubes. The relationship of tonsillectomy to poliomyelitis is discussed.

Birdsall enumerates and evaluates signs and symptoms commonly regarded as reasons for removing tonsils and adenoids. Many of these symptoms attributed to diseased tonsils and adenoids are really due to sinusitis.

Penicillin therapy has great possibilities as a cure of osteomyelitis of the frontal bone and perhaps even of cavernous sinus thrombosis.

Fox reports excellent results in the treatment of epistaxis by intramucosal injection of a sclerosing agent.

Eosinophilia in polyps and in nasal smears is an indication of allergy, although its absence does not rule out allergy.

Hilding has made observations on the manner of the natural drainage of the sinuses.

The relationship of the lymphatics to diseased sinuses is discussed.

Graves and Edwards discuss in detail the function and the structure of the eustachian tubes. The pharmacology and the effects of various shrinking solutions on the nasal membranes have been studied by Lierle, Evers and others.

Pastilles of penicillin have apparently helped local infection of the mouth and throat.

There has been nothing new of note recently in the treatment of atrophic rhinitis.

Some surgical procedures are discussed. Eisenstodt believes that conservative submucous resection might often be used in children, with no interference with the subsequent development of the nose.

Nasal trauma is commented on. Imperatori describes a Mackenty operation on a child with atresia of the pharynx following tonsillectomy and adenoidectomy.

Several reports of tumors are mentioned.

HITSCHLER, Philadelphia.

Miscellaneous

TOTAL HERPES ZOSTER OF THE OPHTHALMIC, MAXILLARY AND MANDIBULAR DIVISIONS OF THE TRIGEMINAL NERVE. HUGH O'NEILL, Arch. Ophth. **33**:237 (March) 1945.

Herpes zoster ophthalmicus, or herpes zoster of the gasserian ganglion, is rare. Indeed, it is so rare that Rice stated that no cases of ophthalmic herpes zoster were mentioned, for example, in the report of one of the largest ophthalmologic and otologic hospitals in the United States, in which over 100,000 patients with ocular disease were treated in 1922. On the other hand, Gundersen, in searching for cases of herpes zoster ophthalmicus, found 10 per annum at the Massachusetts Eye and Ear Infirmary, in Boston, over a five year period.

Herpes zoster involving the ophthalmic, maxillary and mandibular divisions of the fifth cranial nerve is rare, judged by the absence of reports of cases in the literature. Herpes zoster of the trigeminal nerve with concomitant cutaneous involvement of the geniculate ganglion, the combination of which likewise has not been reported in the literature, is rare indeed, although Fuchs, cited by Graves, reported 2 cases in which with herpes zoster ophthalmicus there was concurrent involvement of the seventh cranial nerve, with resulting lagophthalmos and keratitis. This combination is not rare, nor is that of herpes zoster ophthalmicus with involvement of the third, fourth, sixth and seventh cranial nerves, with ocular palsy.

O'Neill reported a case of partial herpes zoster of the seventh cranial nerve, involvement of the vestibular ganglion of the eighth cranial nerve and complete herpes zoster involving all three divisions of the trigeminal nerve. On account of the severe pain, the ocular involvement and the residual scarring on the cutaneous branches of the trigeminal nerve, a diagnosis of herpes simplex or of herpes febrilis was inadmissible. The ocular signs included optic neuritis, exophthalmos, keratitis profunda and probable mild involvement of the third, fourth and sixth cranial nerves. No evidence for classifying this case as one of symptomatic herpes zoster arising from encephalitis, syphilis, tuberculosis, leukemia, Hodgkin's disease, hemorrhage in the cerebellopontile angle or cerebral tumor was found. On the other hand, in view of the severe pain and the cutaneous herpetiform manifestations the case was obviously one of idiopathic herpes zoster of the fifth (complete), seventh (partial) and eighth (vestibular) cranial nerves.

SPAETH, Philadlephia. [ARCH. NEUROL. & PSYCHIAT.]

RELIABILITY OF INFANT SPEECH SOUND DATA. ORVIS C. IRWIN, J. Speech Disorders **10**:227, 1945.

Irwin suggests that studies of infant speech sound data may be improved by statistical study. First, rank-order correlations were obtained from a sample of 1,047 records of speech sounds of infants from 1 to 30 months of age. Two records were selected per month, 60 in all. Each record includes the sounds recorded during 30 expirations of a given infant. The results show the consistency of the sample randomly drawn. Second, the method of percentage of agreement offered a more detailed analysis of the reliability of the data. Third, adequacy of the size of the sample by range of value and number and percentage of value is greater than 0.70. Another sample was made by selecting 64 records ranging from the third to the thirtieth month. Satisfactory reliabilities were obtained by these various methods.

PALMER, Wichita, Kan.

DIAGNOSIS AND TREATMENT OF 72 CASES OF HOARSE VOICE. ARLEIGH B. WILLIAMSON, Quart. J. Speech **31**:189, 1945.

Forty-five men and 27 women were examined because of their hoarse voices. Only 3 of them showed definite pathologic changes of the vocal cords. Hoarseness is therefore primarily a vocal problem, not a laryngologic problem. The most common principal cause was tension of the throat resulting from an effort to speak at a level far below the optimum pitch. Placing the voice in an efficient pitch and removing tension of the throat generally remedied the hoarseness. A common cause of abnormal pitch was a tendency to confuse low fundamental pitch with the lower vocal frequencies usually attributed to bass, baritone and contralto voices. The highest percentage of instances in which a complete remedy was obtained was accomplished in 2 college terms of instruction. The conclusions are somewhat divergent from those usually found in texts on speech correction.

PALMER, Wichita, Kan.

RHINOPLASTIC CORRECTION OF COMMON NASAL DEFORMITIES. J. H. BARRETT, Texas State J. Med. **41**:315 (Oct.) 1945.

Barrett considers that in spite of the length of time that plastic surgery of the nose has been practiced, many rhinologists neglect operations that are indicated. He describes his methods for the correction of (1) hump nose, (2) elongated nose, (3) bulbous nasal tip and (4) deflected nose. For deformities of the septal cartilage he prefers Seltzer's method of elevating the perichondrium and removing a strip of the cartilage at the point of convexity, suturing the cartilage and keeping the septum in position until healing occurs by means of a nasal splint. Where the deflection involves the bony septum, he used Sparer's method of removing an angular wedge from the broad concave side. Metzenbaum's method of cross-hatching the cartilaginous septum is also employed, also Joseph's method of removing a wedge-shaped segment from the septal cartilage. Blair's method of freeing the septum and anchoring it to a bicuspid tooth is described. Consideration is also given to Griesman's method of separating the skin from the underlying bone or cartilage, as near to them as possible in order to avoid undue formation of scar tissue, which interferes with nasal muscular action and so alters facial expression.

SELTZER, Philadelphia.

THIRTY-FIVE CASES OF OTOGENIC FACIAL PALSY IN WHICH THE NERVE WAS OPERATED ON AFTER THE METHODS OF BALLANCE AND DUEL AND BUNNELL. KARSTEN KETTEL, Nord. med. **24**:1783 (Oct. 6) 1944.

Operations on the facial nerve (decompression, nerve grafting, nerve suture) must as far as possible be performed on the site of the injury, for only in this way can the emotional movements of the patients be restored.

In choosing between operation and expectation the faradic reaction is no reliable guide. If it is positive, it suggests a good prognosis; if negative, only that the issue is doubtful, and in some cases the negative reaction also is followed by complete spontaneous recovery.

On the basis of a reexamination of 169 patients who were conservatively treated for facial palsy, the author has previously (Facial Palsy of Otitic Origin, ARCH. OTOLARYNG. **37**:303 [March] 1943) roughly outlined his opinion as to which groups with otogenic facial palsies ought to be treated conservatively. The faradic reaction is resorted to only if there is any doubt.

For otogenic facial palsies the author recommends the following procedures:

I. Preoperative palsies: (*a*) Palsy occurring in otitis media acuta suppurativa. This palsy is of a toxic nature. When it occurs at an early stage of the disease it does not in itself indicate simple mastoidectomy, let alone decompression, for either the aural disease or the future function of the nerve. Mastoidectomy is

indicated only when the palsy is constantly increasing despite a free outlet through the tympanic membrane, or at least does not decrease when observed for some time. If the palsy occurs at a later stage of the illness, however, it indicates simple mastoidectomy, which will then as a rule also be necessary for other reasons. Decompression should not be performed at the same time, as in most cases by far the palsy will disappear spontaneously after the mastoidectomy; only in the rare cases in which after some time spontaneous regeneration has not taken place should decompression be done, provided that the faradic reaction is negative. When the labyrinth is involved, the facial palsy as such does not indicate labyrinthectomy, the number of cells in the spinal fluid being the all-important factor.

(*b*) Palsy occurring in otitis media chronica suppurativa. Here palsy is usually due to granulating inflammation affecting the facial nerve, and in such cases a radical operation is always indicated. When a fistula of the facial canal is demonstrated, the nerve should be decompressed and the nerve sheath split. If no fistula is found, expectation is justified, but if the palsy does not disappear after some time and the faradic reaction is negative (implying a doubtful issue), decompression is indicated. If the continuity of the nerve is broken, nerve grafting or nerve suture should be performed, according to circumstances.

II. Postoperative palsies: 1. Palsy arising in direct connection with the operation. In most cases this is of traumatic origin.

(*a*) Simple mastoidectomy. With conservative treatment the prognosis is bad; in 57 per cent of cases the result was permanent, complete paralysis. There must be immediate decompression in order to establish the nature of the injury. In 5 cases the continuity of the nerve had been interrupted, and the diastasis was extensive. In 3 of these nerve grafting was performed (cases 1 to 3), in all of which facial mobility was recovered; in the 2 remaining ones nerve suture was done (cases 4 and 5), in only 1 of which the treatment was successful; in the other, a case of paralysis of five years' standing from osteitis tuberculosa, it was a failure. In a further 3 cases (6, 7 and 8) the nerve was seriously damaged, but its continuity was not entirely interrupted. In case 7 decompression plus neurolysis gave a successful result, in cases 5, 6 and 8 decompression plus nerve grafting was done. One patient was cured; the other, however, recovered only slight mobility, though the paralysis was not noticeable when the face was at rest (case 8).

(*b*) Radical mastoidectomy–labyrinthectomy. Treated conservatively, 60 per cent of the patients will have permanent, complete facial paralysis. Here, too, immediate decompression is indicated. In 12 patients with broken continuity of the nerve 9 graftings were performed, 8 of which were successful (cases 9 to 17), and 3 nerve sutures (cases 18 to 20), with good results. In 8 others the paralysis was due to a fistula on the fallopian canal (cases 21 to 28); decompression restored full facial mobility in 5 of them and partial mobility in 3. In 1 patient the fallopian canal had become fractured, and a fragment of bone had pinned the nerve; this was removed, and the function was partially restored (case 29).

In all cases of complete facial palsy arising in direct connection with the operation, decompression, nerve suture or grafting is immediately indicated.

2. Palsy occurring at the end of a free interval.

(*a*) Simple mastoidectomy. As an interruption of the continuity of the nerve is out of the question, edema or a hemorrhage in the fallopian canal must be suspected. The paralysis will appear within one or two days and will, as a rule, disappear within a couple of weeks. If it does not and if the faradic reaction is negative, the surgeon should proceed to decompression. Paralysis which does not appear until late in the illness is a symptom of osteitis progressing in spite of the first operation and indicates revision. If the faradic reaction is negative, decompression should be undertaken at the same time. Two such cases are reported (cases 30 and 31).

(*b*) Radical mastoidectomy–labyrinthectomy. The most frequent cause is pressure from a tamponade, on the removal of which the paralysis will disappear

spontaneously. Another cause is hemorrhage in the fallopian canal. In 2 patients, in both of whom complete facial palsy set in after a free interval of a few hours, the faradic reaction was negative seventy-two hours later (no tamponade applied, no fistulas on the fallopian canal). In one of them (case 32) decompression was not performed until three months later, and not until seven months after the decompression did facial mobility begin to return; faint traces of the paralysis still remain; in the other (case 33) decompression was performed immediately, and the paralysis disappeared completely in the course of four and a half months. If, therefore, facial palsy appears at the end of a brief free interval following a radical mastoidectomy–labyrinthectomy, and pressure of a tamponade may be regarded as excluded, decompression should be performed, provided the faradic reaction is negative. A late onset of the palsy is a symptom of osteitis progressing in spite of the first operation and indicates immediate revision.

Many facts tend to prove that postparalytic contractions are due to chronic irritation around the nerve in the fallopian canal. In case 35 the contraction disappeared immediately after decompression; the patient did not show even transient paralysis and did not reappear later (observation period, two and a half years).

News and Comment

FIRST PAN-AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

Immediately following and overlapping with the annual meeting of the American Academy of Ophthalmology and Otolaryngology in October of this year, the First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology will take place, under the sponsorship of the Academy. This congress will open with a banquet and evening session on Thursday night, October 17, and will continue on the two subsequent days. The first scientific session will be a joint session with the otolaryngology section of the Academy, and three additional sessions will take place on Friday afternoon, Saturday morning and Saturday afternoon. All interested members of the Academy are strongly urged to plan on remaining for the Pan-American Congress, for which an excellent program is being arranged.

The registration fee will be $5.

EXECUTIVE COMMITTEE, PAN-AMERICAN CONGRESS
GORDON B. NEW, M.D., *President*
CHEVALIER L. JACKSON, M.D., *Secretary*
PAUL H. HOLLINGER, M.D., *Treasurer*

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

Predident: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Palmer House, Chicago. Time: Oct. 17-19, 1946.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va
Place: San Francisco. Time: July 1-5, 1946.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 13-18, 1946.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Drake Hotel, Chicago. Time: May 31, 1946.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Place: Drake Hotel, Chicago. Time: May 29-30, 1946.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary. Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Drake Hotel, Chicago. Time: May 27-28, 1946.

SECTIONS:

Eastern.—Chairman: Dr.Philip E. Meltzer, 20 Charlesgate West, Boston 15.
Southern.—Chairman: Dr. E. Trible Gatewood, Professional Bldg., Richmond 19, Va.
Middle.—Chairman: Dr. Lawrence R. Boies, Medical Arts Bldg., Minneapolis 2.
Western.—Chairman: Dr. Clarence A. Veasey Jr., Paulsen Bldg., Spokane 8, Wash.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Time: May 31-June 1, 1946.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.
Place: New York: Time: Nov. 14, 1946.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.